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APRIL, 1909

QUANTITATIVE PHARMACOLOGICAL STUDIES:

ADRENALIN AND ADRENALIN-LIKE BODIES

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By

W. H. SCHULTZ

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QUANTITATIVE PHARMACOLOGICAL STUDIES—ADRENALIN AND ADRENALIN-LIKE BODIES.[a]

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The active principle of the adrenal gland, like some of the other internal secretions, is unique in its action. It is supposed to fulfill a function both in normal and in pathological conditions of the human body that makes it a substance of the greatest interest. But aside from the importance that attaches to it because of this, adrenalin has come to hold a place in therapeutics which, until recently, could not be filled by any other known compound. Soon after the discovery of its physiological action, adrenal extract came into extensive use as a styptic and, to a more limited extent, for other purposes. With the discovery of methods for the isolation of the pure principle of the gland, a still wider field of usefulness was found for it as a drug, whereupon numerous preparations were placed upon the market and manufacturing firms widely advertised the importance of the pure product. The different preparations, however, varied greatly in physiological activity—some even being worthless, this being due partly to a lack of care in the process of preparation and partly to the nature of the container and solvent used in bottling the extract. Intravenous injections of the active principle have been suggested in cases of surgical shock, and for such it is a matter of the greatest moment that the solution be of a known concentration. Obviously, too weak a preparation would fail to produce the desired effect and too strong a solution might not only throw too great a strain upon an already weak heart, but cause an after depressing effect no less dangerous.

It is evident, therefore, that every container should be so stamped as to indicate not only the actual strength of the solution in terms of pure base, but it should be standardized pharmacologically against a preparation of known purity, and each package so labeled and dated. At least this should be done with solutions intended for

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purposes of injection, so that the physician or surgeon might be insured against the possibility of securing a solution of unknown strength.

Deteriorated solutions and preparations made from the natural base of uncertain purity have not been the only sources of trouble. Not long since, synthetic dl-adrenalin was placed upon the market and the most positive claims made in its behalf. It was represented as having even a little more pronounced action than natural l-adrenalin, and these claims were supported by what seemed to be researches by expert pharmacologists. Herein will be found data which fail to coincide with the claims made for dl-adrenalin. The investigation has been carried on in view of the fact that physiological methods are necessary in assaying adrenalin-like bodies. A comparison of the relative values of various methods has also been made, together with an effort to improve if possible these methods.

HISTORICAL.

The early discovery of the vaso constrictor action of the adrenal extract by Oliver and Schäfer (61), and by Szymonowicz and Cybulskiego (70 and 71) introduces an epoch in physiological science, the development of the chemistry and physiology of which is scarcely less interesting than that represented by the modern theory of physiological oxidations.

Abel and others, by their extensive chemical research, opened up the way for the discovery of the active principle which was finally separated by Takamine and by Aldrich. Soon after enough data had accumulated to warrant an attempt at synthesizing adrenalin. Stolz succeeded in making the dl-product which was found somewhat different from the natural base, and, finally, Flächer split the dl-compound into its dextro- and levo- components, the latter of which is said to be identical in every respect with the levoadrenalin obtained from the adrenal gland.

The physiology of the adrenal glands alone covers a field of no mean proportions, and it is more or less closely associated with a still larger number of articles on the therapeutic and toxic action of adrenalin. Since, however, the studies to be described are for the most part quantitative in character, it seems advisable to limit references as far as possible to methods and results that are more or less quantitative which deal with the physiological action of adrenalin and closely allied compounds. Comprehensive résumés of the literature dealing with the adrenals, adrenalin and its homologues, may be found in the excellent papers by Hultgren and Anderson (40), Abel (2 and 3), Aldrich (4), Möller (58), Davis (22), Vincent (74), Rolleston (64), and Schäfer (65).

The early anatomists observed that the juice of the medullary substance of the *capsula atrabiliana* (suprarenals) darkened upon exposure to the air, and called it *atra bilis*. It was not, however, until the nineteenth century that light was shed upon the cause of this color change. In 1856 Vulpian (75) observed that the medullary juice of the suprarenals turned emerald green and rose carmine when brought into contact with ferric chloride and iodine, respectively. These reactions were characteristic of this organ, and at once led to the surmise that the medulla of the gland contained a substance of physiological importance.

Pellacani (62) as early as 1879 performed in Foa's laboratory a series of very interesting experiments with extracts of the fresh organs, among which was a series of injections of adrenal extract into various animals. The capsules were excised, ground up in a mortar, and to this mass was added distilled water sufficient to filter, but not exceeding the amount needed for the injection. This liquid extract was filtered through linen and then paper, after which it was immediately injected. The greatest care was exercised both in the making of the extracts and in the preparation of the instruments, the latter being immersed in strong alcohol. Subcutaneous injections were made into dogs, cats, rabbits, guinea pigs, and frogs. A rise of temperature did not always occur, and in some of the animals there was a lowering of temperature, followed by death in from twelve to thirty hours for the more rapid cases of intoxication. The symptoms were general excitement, followed by paralysis, both sensory and motor, increasing weakness of the heart, lowering of temperature, 4 5°, followed by death. It is strange that Pellacani should have secured such characteristic adrenalin effects and yet observed that subcutaneous injections were more toxic than intra-peritoneal ones, and that both of these methods of injection caused more pronounced and quicker effects than intravenous injections. Although he used extracts of organs other than the adrenals, he found the latter to be more toxic and the following organs to be progressively less active: Muscle, liver, kidney, brain, milt. Five grams of clear extract of two suprarenals of rabbits injected into a 770-gram guinea pig caused death after twelve hours. Eighteen to twenty cubic centimeters of extract of cat or guinea-pig adrenals proved very toxic for rabbits, whereas a higher dose, 1 gram, of lamb or heifer suprarenal extract was necessary to cause a like degree of toxicity.

Mattei (53) repeated Pellacani's experiments, but arrived at very different conclusions. In his conclusions he states that water extracts of fresh organs injected into different animals does not cause any toxic action. The animals that die do so because of the after effects of the soluble organic matter of the decomposed tissues, which later cause septicemia.

Foa and Pellacani (32) in 1883 confirmed Pellacani's earlier results and removed, as they supposed, the influence of fibrin ferment by heating the extracts to 60°. They found the toxic substance to be very soluble in water and alcohol, but rather insoluble in ether and chloroform. Whereupon they purified the water extract as follows: The capsules were minced and put in boiling water; after a time the water was decanted, evaporated to dryness, and the residue treated with alcohol, the almost colorless alcohol solution evaporated to dryness and again taken up with water, thus leaving behind most of the impurities. Upon evaporating this solution a dark residue of a peculiar odor and of very acid reaction was obtained. One gram of this substance injected into dogs caused death, whereas the extracted pulp of the capsule was relatively inert. These investigators came to the conclusion that there is an active poison in the adrenal gland independent of fibrin ferment which causes extreme prostration, collapse, motor and sensory paralysis, and death from asphyxia because of paralysis of the medulla oblongata, especially the respiratory center. After examining the original articles of these early writers it seems that more credit should be given to their work than is usually found in literature, for they certainly described symptoms of poisoning very characteristic of adrenalin.

Krukenberg (44) (1885) assumed that the substance giving color with ferric chloride is not the chromogenic substance of Vulpian, but more likely pyrocatechin accompanying the chromogen.

Marino-Zuco (52) (1888) after freeing 50 capsules from fat, ground and macerated them in 1,000 c. c. of distilled water heated in a water bath for several hours. The mixture was strained, evaporated on a water bath, and filtered, the resulting solution being slightly acid and red in color. This extract, diluted to 200 c. c., and 1 c. c. injected subcutaneously into rabbits, caused death in five minutes. If, however, the extract were treated with acid or alkali, it was ineffective, even in large doses. The presence of neurine alone does not explain the toxic action of the pure extract, but in combination with very acid phosphates it may, at least when made artificially, prove very toxic.

Guarnieri and Marino-Zuco (35) (1888) made an extract of 10 beef suprarenals in 60 c. c. of water and injected 1 c. c. into medium-sized rabbits, causing death in a very short time. Treatment of the extract with acids and other reagents lessened the activity of the extract when intravenously injected. From these results they concluded the toxicity of the aqueous extracts was due primarily to neurine (which I believe is now generally conceded to have been choline) and organic phosphates.

It is interesting to know that Dzierzgowski (27 and 28) as early as 1893 synthesized a number of catechol derivatives closely related to the ones discussed in this paper. The work, however, did not attract much attention at the time and seems to have been lost sight of, their physiological activity not having been tested by him. Even Stolz, who was familiar with the work, does not give Dzierzgowski and his contemporaries the credit they deserve for their pioneer work with these compounds. Among some of the compounds synthetized by Dzierzgowski are dimethyl-amido-aceto-catechol, anilido-aceto-catechol, methyl-anilido-aceto-catechol, quinolin-, pyridin-, and piperidin- aceto-catechol, aceto-chloro-aceto- and chlor-propio-catechol.

Gluziński (34) (1895) removed aseptically the adrenals of recently killed oxen, calves, hogs, rabbits, and dogs; weighed and ground them up with broken glass. One part of the pulp was allowed to macerate eight to twelve hours in a cold 50 per cent aqueous solution of glycerine. The solution was filtered through sterilized glass wool and the clear extract injected from a sterilized syringe into the ear vein of a rabbit. In one minute 0.3 to 1 gm. killed a 1,500 gm. rabbit. The extract heated to 100° for one hour retained its toxicity, causing hemiplegia, loss of sensibility, cramps of anterior part of body, opisthotonus, rapid breathing, dilation of the pupil, and, finally, symptoms of dyspnœa, general paralysis, and, unless artificial respiration were administered, death from asphyxia.

Moore, B. (59) (1895), basing his opinion on experiments which indicated that such chemical operations as destroy the color reactions by oxidizing the reducing agent likewise alter the physiological activity of the extract, concluded that Vulpian's chromogen and the active principle are identical.

Dubois (25) (1896) used an extract of fresh suprarenals of rats ground in equal volumes of alcohol and distilled water and allowed to remain twenty-four hours in glycerine. He concludes that such extracts contain two substances, one soluble in alcohol at 90°, causing vaso-dilation and congestion; the other, very soluble in this reagent, causing paralysis, depression of the heart, and death from asphyxia.

Fränkel (33) (1896) separated what he supposed to be the active principle, calling it "sphygmogenin," and observed that a close relation existed between the ease with which the substance is oxidized and its physiological activity. He suggested that "sphygmogenin" is a pyrocatechin derivative, and although he and Krukenberg were in a measure correct it is now known that Fränkel's product was a mixture.

Vincent, S. (73) (1897), performed a series of 80 experiments with rabbits, guinea pigs, rats, mice, frogs, and toads. The extract was made by boiling a short time and filtering the chopped-up gland, although a dried gland, glycerine, or alcoholic extract was sometimes used. Five-tenths gram of the fresh gland in the form of a glycerine extract injected into the dorsal lymph sack of the frog caused immediate paralysis, from which the animal soon recovered, doses up to 3 gm. not proving fatal. With toads much larger doses were required to produce corresponding symptoms. Doses equivalent to 1.5 and 3 gm. of fresh gland caused in rats the usual cardiac, respiratory, and nervous symptoms, some of the animals recovering, others dying from failure of respiration. Guinea pigs of average weight were injected with an equivalent of 6 gm. of fresh gland, the symptoms being practically the same as for cats and mice, except that the urine of the former was more blood colored (with or without corpuscles). The fatal dose for rabbits can not be stated, since they vary so much in reaction. Unlike the guinea pig, which becomes very restless, the rabbit grows drowsy and listless. Small initial doses followed by one that was usually fatal for fresh rabbits no longer proved to be so. Vincent thinks that a partial immunity (tolerance) is established toward the toxic action of the extract, which passes off after a few weeks.

In the meanwhile Abel (1899), Von Furth, and others had been working upon the chemistry of the active principle. The former isolated a substance which he called "epinephrin;" the latter a substance which he called "suprarenin." A controversy arose as to which was the active principle. As a matter of fact, neither chemist seemed to be working with the pure substance. They did, however, throw much light upon the chemistry of the compound, and prepared the way for its separation by Takamine (72) (1901) and by Aldrich (4).

Moore and Purinton (60) (1899) criticized the idea of epinephrin (Abel), suprarenin (Von Furth), and the other so-called pure products being the active principle. They record a rise of blood pressure after intravenous injections of the crude medullary extract in doses ranging from 0.245 to 24 millionths of a gram per kilo. This, they maintain, represents a physiological activity far in excess of any of the so-called active principles.

In reply to Moore and Purinton's criticism, Hunt (41) determined the minimal amounts of Abel's epinephrin sulphate necessary to cause a rise of blood pressure, finding that even so small an amount as 0.083 millionths of a gram per kilo body weight caused a rise of 5 mm. of mercury, and 0.23 millionths of a gram per kilo, a rise of 7 mm. The duration of the injection period (in this case two to five seconds) was found a very important factor in determining the degree of vaso-constriction resulting from a given dose of adrenalin. One and one-tenth millionths of a gram per kilo injected rapidly might cause a rise of blood pressure equal to 14 mm. of Hg., whereas double this dose injected slowly caused a rise equivalent to but 8 mm.

of Hg. Hunt maintains that these results justify Abel's contention that epinephrin is the active principle.

Cybulskiego (19) found that 1 c. c. of a 10 per cent solution of the extract injected into the vein of a rabbit caused death, but if this solution were diluted ten to twenty times the same dose of adrenalin was borne without any untoward effects.

Bouchard and Claude (14) (1902) experimented with a small number of animals, finding that the lethal dose may be only 0.5 or 0.2 mg. per kilo, an animal occasionally withstanding an intravenous injection of as much as 1 mg., 2 mg. usually proving fatal. The lethal dose for rabbits therefore lies between 1 and 2 mg. per kilo. Provided there was a gradual increase of each successive dose until a maximum was reached, they could inject as much as 4 mg. per kilo without any untoward effects other than temporary paresis such as is brought on by an initial dose of 1 mg.

Battelli (7) (1902), using a slight modification of Takamine's method, claims to have secured adrenalin even more pure than that obtained by Takamine. Battelli (9) injected this preparation subcutaneously into rabbits, guinea pigs, and frogs with the following results: With a corresponding number of guinea pigs 10 mg. per kilo proved to be the lethal dose. On the other hand, frogs were ten times more resistant than rabbits, 1,000 mg. killing only three out of four.

Dose per kilo.	Number of rabbits injected.	Number of animals died.
Mg.		
2	5	0
10	6	5
20	3	3

He also (8) (1902) notes that Gluziński in 1895 called attention to the fact that intravenous injections were more toxic than subcutaneous ones. Battelli experimented with what he considered a very pure product, using adrenalin base dissolved in water acidulated with hydrochloric acid and neutralized with Na_2CO_3 just before injecting into the femoral vein, and obtained the following results:

Rabbits—0.1 mg. per kilo, not lethal.
0.2 mg. per kilo, 1 out of 5 died.
0.4 mg. per kilo, 3 out of 4 died.
0.6 mg. per kilo, always lethal.
Guinea pigs—0.05 mg. per kilo, not lethal.
0.10 mg. per kilo, 2 out of 5 died.
0.20 mg. per kilo, always lethal.

The toxic dose for the rabbit and guinea pig was about the same when the injection was made into the jugular vein of the rabbit and into the femoral vein of the guinea pig, death ensuing from œdema of the lungs or fibrillation of the heart. Summing up his work, he concludes that intravenous injections are about forty times as potent as subcutaneous ones.

Eeckhout (26) is quoted as finding the lethal dose of adrenalin to be 0.08 to 0.06 mg. per kilo. In the original paper, however, I find that this lethal dose is calculated from doses that accidentally caused death in animals previously injected with morphine and atropine and anæsthetized with chloroform, which, according to later writers, renders the animals less resistant to adrenalin.

Amberg (5 and 6) (1902) compared the toxicity of Abel's epinephrin with the commercial product made by Takamine's method, and found that Abel's sample dissolved completely, whereas only 517.7 mg. of the commercial product dissolved in 18 c. c. of H_2O, leaving behind 4.6 mg. of sediment. After studying the effect of subcutaneous injections upon 9 dogs and intravenous injections upon 16, he concluded that the lethal intravenous dose lay between 0.99 and 2 mg. per kilo, subcutaneous injections of 4.9 mg. per kilo not proving lethal, though 6 mg. or more per kilo were.

Lesage (47, 48, and 49) (1904) does not state by what process his adrenalin was made, but judging from the size of the lethal dose it must have been a very good one. From a stock solution containing 0.04 gm. adrenalin, 40 gm. H_2O, and 1 drop of HCl he made a 1:20,000 solution to be used for intravenous injection.

Rabbits—0.05 mg. per kilo, signs of intoxication.
0.20 mg. per kilo, lethal (4 animals).
Dogs—0.05 mg. per kilo, not toxic.
0.12 mg. per kilo, sometimes lethal.
0.20–0.25 mg. per kilo, usually lethal (4 out of 6).
Cats—0.50–0.81 mg. per kilo, lethal (6 animals survived after 5 injections of 0.25 mg. and 1 of 0.50 mg. per kilo).

With the larger doses, 0.26 mg. per kilo, he observed that the dogs usually died from asphyxia, but when 0.20 mg. per kilo was fatal they usually died from heart failure. In general, anesthetics augmented the toxic action and sublethal doses of adrenalin rendered the animals resistant to doses that ordinarily proved fatal for normal animals. He concludes that there is a considerable variation in the susceptibility of one individual as compared with another, and a still greater variation for different species.

Baylac, J. (10) (1905), working with a 1:1,000 solution of adrenalin, found that the lethal dose varies with the species and according to the manner of its injection. The lethal subcutaneous dose as determined on 6 guinea pigs and 6 rabbits is estimated at 100 mg. per kilo and 20 mg. per kilo, respectively. On the other hand, he estimated from experiments with 5 rabbits that the lethal intravenous dose is about 0.06 mg. per kilo, although 0.03 mg. per kilo may prove very toxic. An intrapleural injection of 2 mg. per kilo or the same amount injected intraperitoneally into guinea pigs may prove fatal.

Up to this time toxicity experiments and blood pressure determinations were primarily qualitative in nature, yet they furnish a most reliable index of the relative purity and activity of adrenal extracts, and of the pure products of the active principle thus far made.

Houghton (38), however, in 1901, emphasized the usefulness of quantitative determinations by the blood-pressure method and published records illustrating the relative effect of different volumes of the same solution injected subcutaneously. In 1902 (39) he proved this method one of the most accurate means of assaying adrenalin. Three solutions, A, B, and C, were used, one containing 85, one 40, and another 130 per cent of adrenalin in a given solution. An assistant reported that A contained 80, B 40, and C 135 per cent of the amount in the known solution. It was also found that Takamine's crystalline product was from 600 to 800 times as active as the best aqueous extracts of freshly prepared glands.

Läwen (46) in 1903 studied the quantitative effect of adrenalin upon the blood vessels of frogs. The brain and spinal cord were destroyed and, by measuring the outflow from the vessels subjected to a given pressure with a known solution, he determined the relative vaso-constrictor action of the different adrenalin solutions. With a pressure equivalent to 30 c. c. of H_2O the frogs lasted about two hours. Two ten-thousandths of a milligram of suprarenin in a concentration of 2:100,000,000 constricted the blood vessels of a 50-gram frog so that the outflow was reduced 20 to 37 per cent. In other experiments it was determined what pressure in excess of the minimum was necessary to overcome the increased resistance due to vaso-constriction. By these experiments Läwen not only could study solutions quantitatively, but could even detect the differences between fresh solutions and those that had been standing for some time.

Up to this time little attention had been given to the possibility of using particular organs for assaying adrenalin, but observations made by the following writers eventually led to the idea that possibly the eye or separate strips of muscle might prove serviceable.

In 1904 Meltzer and Auer (54), Lewandowsky (50), Boruttau (13), and Langley (45) confirmed Foa's observation that adrenalin extract intravenously injected causes dilation of the pupil and that subcutaneous injections have no effect. It is assumed that the extract is oxidized in the lymph spaces before it reaches the neuro-muscle apparatus and effects dilation. Meltzer (51) states that his extensive experience with subcutaneous injections of adrenalin in normal rabbits warrants the conclusion that dilation does not occur unless the dose is large enough to cause asphyxia. If, however, the superior cervical ganglion is excised, even a moderate dose, 0.6 c. c. of a 1:1,000 solution, will cause the pupil on the side from which the ganglion is removed to become dilated *ad maximum*. Radziejewski (63) was one of the first to claim that adrenalin instilled into the eye exerts no effect upon the pupil, and this idea is supported by Lewandowsky (1889), Boruttau, Meltzer, Loewi, Ehrmann, etc. Meltzer (55), however, finds that removal of the superior cervical ganglion and instillations of adrenalin twenty-four hours later made the pupil dilate in proportion to the amount of adrenalin instilled. After four or five instillations, two drops every two or three minutes, the dilation can be *ad maximum*, lasting several hours in rabbits and cats (54).

Meltzer and Auer (56) in a later series of experiments showed that adrenalin subcutaneously injected into frogs or instilled into the eye dilated the pupil widely, the vertical axis being affected most. Three drops of a 1:1,000 solution instilled by pushing a fine pipette between the bulbus and the lid caused very marked dilation in three to seven minutes, which lasted as long as if subcutaneously injected. To eliminate the possibility of the drug being absorbed by the skin the bulbus was excised and adrenalin dropped upon the corneal surface. There was prompt dilation, which lasted many hours. As a result of this work the writers suggested that "the frog's eye excised or *in situ* might prove to be a better reagent than the blood pressure to demonstrate the efficiency of a suprarenal preparation."

Wessely (76) (1905–6) reinvestigated the action of adrenalin upon the intraocular pressure and upon the pupil. He found that instillations of adrenalin into the eyes of vertebrates causes dilation, provided the strength of the solution is increased to suit the animal experimented with. For man a 1 per cent solution is accompanied by danger, and a 0.1 per cent solution is too weak to cause mydriasis.

Schultz (66) also showed that adrenalin instilled into the eyes of mammals always causes dilation of the pupil. The degree of mydriasis resulting from a given amount of adrenalin depends, however, upon the intensity of light stimuli and upon the kind of animal used. There is, so to speak, a kind of antagonism between the processes set up by the light stimuli and those initiated by adrenalin. So that animals with a sensitive and more highly developed light-accommodating mechanism require a longer period of instillation and a greater amount of adrenalin to cause mydriasis than those with eyes less sensitive to light.

Ehrmann (29) (1905) used the method suggested by Meltzer (56), and thinks to have ruled out the influence of the sympathetic impulses, the presence of which is held to prevent mydriasis. The enucleated frog's bulbus placed in a small vessel of 5 c. c. capacity with a known amount of adrenalin was compared with controls in physiological saline. He mentions that there is considerable individual variation, but in the subsequent paragraphs and the reviews of his article this factor is lost sight of. The pupil dilated to a maximum in a solution of 0.001 mg. and 0.0001 mg. per c. c. produced distinct dilation. Having, as he thinks, proved the delicacy of the test object, he then determines the adrenalin content not only of the blood after an intravenous injection of adrenalin, but even determines that the adrenals secrete into the blood a perceptible amount of adrenalin. He overlooks, however, the fact that in the blood there are other substances that cause the pupil to dilate, not to mention certain factors discussed later which enter in to justify the severest criticism of his technique and conclusions.

Meyer (57) (1906), by suspending strips of beef's subclavian and carotid arteries in adrenalin solutions, concludes that with:

1 gm. of adrenalin to one thousand million c. c. of oxygenated Ringer, contraction may occur.
1 gm. of adrenalin to one hundred million c. c. of oxygenated Ringer, contraction usually but not always occurs.
1 gm. of adrenalin to fifty thousand c. c. of oxygenated Ringer, maximal contraction usually occurs.
1 gm. of adrenalin to one hundred c. c. of oxygenated Ringer, maximal contraction may occur.

He maintains that the method is a quantitative one, and along with other interesting statements says that strips exposed to rather concentrated solutions of adrenalin (1:10,000) for eight minutes, removed, wiped dry, and then hung in 20 c. c. of fresh Ringer solution for five minutes diffuses sufficient adrenalin into the new solution to stimulate fresh strips quantitatively equivalent to a one to twenty million solution.

By this time sufficient chemical data had accumulated to warrant attempts to synthesize adrenalin. Stolz (68) and Dakin (21) succeeded, independently, in making substances closely allied to this product, among which proved to be dl-adrenalin. The discovery of these interesting compounds resulted in a new series of pharmacological experiments.

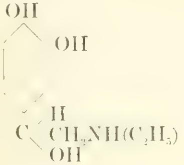
The physiological testing of synthetic substances by Dakin (21) and by Loewi (51) and Meyer are hardly quantitative in nature, and their results, though roughly comparable with each other, give only a general idea of the activity of the compounds. Biberfeld and German writers in general seem, as pointed out, to have been misled by some of the statements of Loewi and Meyer, based upon quali tative rather than quantitative studies of such compounds as aminoketone, ethyl- and methyl- aminoketone, and ethyl- and methylaminoalcohol. Dakin (20, 21) examined analino, o-toluidino, and α-naphthylamino-acetylcatechol, finding that small quantities cause no rise of blood pressure; piperidine does, though its compounds are less active than piperidine itself. He arrives at the generalization that the catechol nucleus is necessary to produce a physiologically active substance of the type of adrenalin; that it is of importance that the hydrogen atoms of both hydroxyl groups in the catechol nucleus be unsubstituted. He thinks also that an alkyl group of low molecular weight attached to the nitrogen atom tends to produce a more active substance than when an aromatic group is attached, whereas derivatives of piperidine, heptylamine, and benzylamine occupy an intermediate position, and, finally, that there seems to be a close connection between chemical instability and physiological activity, and *vice versa.*

One of the first and most strikingly practical applications of quantitative determinations of adrenalin found in literature is that of Hunt (42) (1906). Samples were sent to the Hygienic Laboratory for the determining of their relative physiological activity. He found that the rise of blood pressure from equal amounts of samples A and B were practically the same, about 1.4 to 1.8 times that of C; in order to record the same rise of blood pressure from equal volumes of C and of A and B it was necessary to increase the strength of C by about 1.7 times. In a second series four preparations were tested; three of these were labeled "1:1,000 solutions of the active principle" and one "dried powdered gland." Preparations C and A, from the manufacturers of the first series, were about equal in strength and approximately five times as strong as D. It was possible to prepare from an ounce of the dried suprarenal gland about 15 fluid ounces of a decoction as potent as the active principle labeled 1:1,000. To further emphasize the accuracy of the blood-pressure method I quote an interesting paragraph from the same paper: "Abel calculated that fresh beef's suprarenals contain at least 0.3 per cent of the active principle. One part of the dried gland corresponds, according to the United States Pharmacopœia, to approximately six parts of the fresh gland; hence, according to Abel's experience * * * 1 gm. of the dried gland should contain 0.018 gm. of the active principle * * * and should yield about 18 c. c. of a solution corresponding to 1:1,000 of the active principle. As a matter of fact, I found that it yielded about 15 c. c. [from blood-pressure data] of such a solution, and it is improbable that the gland was completely exhausted in my experiments."

Sollmann (67) (1906) also examined a series of commercial suprarenal preparations and found the relative efficiency to vary considerably. At least two samples, *a* and *b*, made by the same firm, were bought on the open market and tested. In order to eliminate all bias the solutions to be tested were made by a second person. The relative efficiency of the original solutions was thus estimated and found to be as follows: 1a = 70, 1b = 70; 2a = 100, 2b = 86; 3a = 86, 3b = 63; 4a = 95, and 4b = 0.

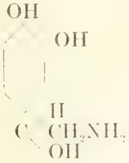
As already pointed out, Dakin and Stolz had synthesized compounds thought to be allied to adrenalin. Biberfeld (11) (1906) compared methylamino alcohol (synthetic dl-adrenalin) with natural adrenalin and found them physiologically identical. A 1:500,000 solution causes dilation of the frog's pupil; 2 mg. injected subcutaneously into a rabbit causes diuresis and glycosuria; 0.001 of a milligram injected intravenously causes a rise of blood pressure. The lethal dose for rabbits is given as 0.1 to 0.2 mg. per kilo for intravenous injections and 4 mg. per kilo when injected subcutaneously.

The reduction product of the aminoketone, ethylaminoalcohol represented by the formula



is said to act more strongly upon the vagus, but to affect the blood pressure in a less degree than natural adrenalin. Diethylamino alcohol was also found to be much less active.

The amino alcohol represented by the formula



produced a rise of blood pressure equal to that caused by natural adrenalin. In one case a rabbit withstood a dose of arterenol two to three times as great as the lethal dose of adrenalin without showing any very untoward symptoms of intoxication.

Gunn and Harrison (36) (1908) dissolved samples of adrenalin in various kinds of media, preserving the 1:1,000 solutions in different kinds of containers and under various conditions, their object being to determine the effect of various factors upon their rate of deterioration. Each of the set of about twenty samples was marked by letter or number and submitted to Dixon for physiological testing. The results when examined by Gunn and Harrison revealed that the fresh samples of natural and synthetic adrenalin, though of the same concentration, did not possess the same physiological activity, the natural product being approximately one-half that of the synthetic. These writers in referring to Dixon's tests conclude that since "the natural substance is lævorotatory and the synthetic is optically inactive, it would appear that only one of the two isomers possesses physiological activity." This is the first published statement of the kind found that is supported by experimental data of so reliable a character, but Dixon (23) in a note in the Pharmaceutical Journal states that "it was Cushny who first suggested at a meeting of the Physiological Society that the synthetic adrenalin might be a mixture of the two optical isomers of which the lævo variety alone was markedly active."

Biberfeld (12) repeated his work of 1906 and not only confirmed to his own satisfaction his earlier results but also criticised unjustly the work of Dixon described by Gunn and Harrison. He weighed equal quantities of the natural and synthetic base, dissolved each in 0.8 per cent sodium chloride solution acidified with the calculated amount of hydrochloric acid, and allowed them to stand for eighteen days when their vasoconstrictor action was tested upon a rabbit weighing 2 kilograms. The rise of blood pressure resulting from intravenous injections of these compounds is given as follows:

0.001 mg. natural, rise of 8.5 m. m.	0.001 mg. synthetic, rise of 7.0 m. m.
0.002 mg. natural, rise of 15.0 m. m.	0.002 mg. synthetic, rise of 11.0 m. m.
0.008 mg. natural, rise of 18.0 m. m.	0.008 mg. synthetic, rise of 19.0 m. m.
0.016⅔ mg. natural, rise of 27.0 m. m.	0.016⅔ mg. synthetic, rise of 27.0 m. m.

This is certainly insufficient data upon which to base such important conclusions, and more especially is this true when part of the data given in itself seems open to criticism. One and five-tenths milimeters difference in such small blood-pressure changes as occur in the first reading and 4 m. m. difference in the second indicate something other than mere experimental error.

Stolz and Flächer (69) (1908), in criticising Gunn and Harrison's article, state that "the two products, synthetic and natural adrenalin are not isomeric substances but identical structurally. The suggestion that synthetic suprarenin is only half as active as the natural lævorotatory suprarenin is incorrect, as otherwise in logical sequence the dextrorotatory suprarenin would be entirely inactive. We made by synthesis a preparation composed of one-half of dextrorotatory and the other half racemic suprarenin. The latter, which is optically inactive, consists of equal quantities of the lævorotatory and dextrorotatory modification, so that this substance consisted of three-quarters dextro and one-quarter lævorotatory suprarenin. If the supposition were correct that the dextro-rotatory suprarenin is inactive, then this mixture could only possess one-fourth the activity of the natural suprarenin. Careful experiments upon animals with the kymograph demonstrate, however, the complete equivalence of this preparation with the natural substance." These writers are not content with this statement, but even make it appear as if a commercial product in which they are deeply interested is, according to "a great number of clinicians and pharmacologists, not only equal to that obtained from the adrenal organs but its activity is even greater than the latter, which must be attributed to the absolute purity of the synthetic preparation."

Cushny (16) (1908), in a preliminary report, states that he examined the products just mentioned, finding that the strength of the three substances—natural base, synthetic base, and the three-fourths dextrorotatory base—bear the relation 4:2:1. He also finds Biberfeld's results divergent from his own, and offers as a possible explanation the use of rabbits which, Cushny thinks, acquire a tolerance for adrenalin.

Cushny (17) (1908) compared a sample of natural adrenalin the optical activity of which was found to be -42.25° with a synthetic sample optically inactive. Instead of trying, as did Biberfeld, the relative activity by comparing the minimal doses necessary to cause a rise of blood pressure he checked 1 c. c. of 1:100,000, 1:50,000, and 1:25,000 of adrenalin base with corresponding solutions of the synthetic product. Then by decreasing the dose of the stronger preparation and increasing that of the weaker until the rises of blood pressure approximated each other he was able to determine the relative physiological activity of each. As a result of his experiments upon dogs anesthetized with paraldehyde with both vagi cut and under conditions of artificial respiration he concludes that the "natural lævoadrenalin acts twice as strongly on the blood pressure as synthetic or racemic adrenalin, and presumably also upon the other organs affected by adrenalin. From this it is inferred that dextroadrenalin is devoid of action on these tissues, and this is confirmed by the examination of a partially isolated d-adrenalin."

Flächer (31) (1908) succeeded in splitting dl-adrenalin into its d- and l- components. He concluded that the physico-chemical properties of the synthetic l-adrenalin and the natural l-adrenalin are identical. Both melt at 211° to 212° C. (uncorrected). They form oxalates and chlorides that do not crystallize. The synthetic product purified from the bitartrate and dissolved in hydrochloric acid shows an optical activity of $[\alpha]_D^{19.6} = -51.40^\circ$ and that of the pure natural l-adrenalin obtained from the bitartrate showed an optical activity of $[\alpha]_D^{19.8}$ 51.40°. The d-adrenalin dissolved in weak hydrochloric acid showed an optical activity of $[\alpha]_D^{19.8} = +51.80$ and, like its optical isomer, melts at 211° to 212° C. (uncorrected), and it forms oxalates and chlorides which do not crystallize.

The physiological activity of these new products was tested by Abderhalden (1) (1908) and Müller, and reported in a very unsatisfactory manner. The l-adrenalin was found to be fifteen times more active than the d-isomer.

Cushny (18) (1908) in a very brief report states that he tested the synthetic d- and l- adrenalin prepared by Flächer, finding the synthetic l-adrenalin equal in potency to the l-adrenalin obtained from glands. It was found difficult to determine the absolute relative physiological activity of the synthetic d- and the synthetic l- adrenalins, but in general ten times more synthetic d-adrenalin was required than of the synthetic l- to produce a given rise of blood pressure. It was calculated that the relative physiological activity of the d- and l- products were in the ratio of 12:1. In the light of these experiments the relative activity of the natural l-adrenalin and the synthetic dl-adrenalin may be expressed by the ratio 24:13.

Although Emmert's work (30) (1908) has to deal primarily with histological changes ensuing from repeated injections of sublethal doses of adrenalin, some interesting toxicological data is recorded. The number of mice used by him does not seem to have been large, and hence the dose given as lethal must not be taken too seriously. It is stated that 0.1 mg. generally caused death in from one minute to sixty hours. In one case he was able to increase gradually the dose to as much as 0.5 mg. before acute poisoning set in. In another case three mice were injected with 0.1 mg. once or twice a day for 9, 24, and 39 days. In still another experiment mice were observed for 109 days, fifty-six 0.033 mg. doses being first injected, and later twenty-five 0.1 mg. doses. Only three mice lived after doses of 0.1 to 0.15 mg., and these were always prostrated by each of the twenty-five injections given in the course of 24 days. For the object of studying lesions caused by repeated injections his experiments are perhaps sufficient, but for purposes of determining the lethal dose they are not.

THE RELATIVE ACTIVITY OF ORTHO-DIOXY-PHENYL-ETHANOL-METHYL-AMIN (NATURAL L- AND SYNTHETIC DL- ADRENALIN), OF ORTHO-DIOXY-PHENYL-ETHANOL-AMIN ("ARTERENOL"), AND ETHYL-AMINO-DIOXY-ACETO-PHENON ("HOMORENON") AS DETERMINED BY BLOOD PRESSURE.

Soon after synthetic dl-adrenalin was placed upon the market samples of it were submitted to the Division of Pharmacology of the Hygienic Laboratory for comparison with the natural l base. A few preliminary tests showed the synthetic substance to be only one-half to two-thirds as active as the natural. Upon noting this, it was decided to make a study of certain catechol derivatives and also to examine into the best methods for standardizing them. Of the several methods proposed it was found that on the whole that of blood pressure, of the pupil, and of subcutaneous injections was most satisfactory. The pupil method as used by Meltzer and by Ehrmann is adequate for qualitative but not for quantitative testing. Hence this method was changed to eliminate as far as possible the most serious errors that could arise, which has now made the pupil method, though not quite so delicate, almost as reliable as that of blood pressure. Likewise the toxicity data of adrenalin literature, though in a general way supplementing the qualitative and quantitative results of their period, are, on the whole, unsatisfactory, being unsuited for comparison with more recent experiments with pure compounds. For this reason a series of experiments was carried on under conditions whereby the members of one series could be compared with those of another.

A glance over the literature on adrenalin reveals at once how prominent a place the blood-pressure method occupies in testing this drug both in a qualitative and quantitative manner, and it would seem to be the most consistent test for catechol derivatives, these substances being primarily vaso-constrictors. Because of this the relative pharmacological action of the compounds already mentioned will first be considered in terms of rise of blood pressure and all subsequent results by other methods will be referred to this as a standard.

One of the first difficulties encountered was the variation in the activity of adrenalin found upon the market. In order to eliminate this factor the Hygienic Laboratory purchased direct from a manufacturing firm 19.4 grams of natural l-adrenalin base. This they claimed to contain 15 grams of pure adrenalin, their basis for calculation being that incineration left 22.6 per cent ash, which, as will be seen later, was erroneously inferred to represent all the impurities present. To make sure of a good preparation, however, the sample was repurified by Taveau, chemist in the Division of Pharmacology, who has done so much valuable work in synthesizing compounds of this character both in Abel's laboratory and in the Division of Pharmacology of the Hygienic Laboratory. He obtained upon purifying the 19.4 grams of adrenalin 5.5 grams of a fine crystalline ash-free base of unusual physiological activity with an optical activity of $[\alpha]_D^{24} = -53.40^{\circ}$.[a] An additional one-half gram of practically ash-free base made a total of 6 grams. There was, of course, some loss in the process of purifying, but that this unusual loss was one due not merely to chemical manipulation but rather to the impurities eliminated is shown by physiological testing. The original sample was checked against the repurified sample and shown by the blood-pressure method on cats to be only one-half to one-third as active. These findings were eventually confirmed by the physiologist of the manufacturers and concurred in by them, as is evidenced by their voluntarily sending a bill for 6 instead of for 15 grams of adrenalin base.

Having secured an unusually pure sample of adrenalin base to be used as a standard, the next problem was to find an anesthetic that would not increase the secondary depressing action of adrenalin upon the heart and yet maintain a constant state of anesthesia. An animal in order to yield a uniform blood-pressure record must of course be so anesthetized as to maintain the irritability of the parts affected by the drug at not too high nor too low a threshold value, at a level where absence of pain is assured and yet where motor disturbances are removed.

It may not be amiss to speak for a moment of anesthetics that under certain conditions seem to fail in these requirements, themselves bringing about results which at first glance might be attributed to the effect of adrenalin itself. Anesthesia from urethane and chloral, chloretone, and paraldehyde of course have advantages, but the stomach puts beyond the control of the operator all subsequent adjustment of the degree of narcosis. And I am inclined to think that in the latter part of long experiments there may be present a condition of too low an irritability, so that small doses of adrenalin at first effective are now no longer so. This condition seemed to be present in cats anesthetized with urethane chloral, but not when under light ether anesthesia. According to Alexander-Lewin animals may be chloralized to the extent of annulling all action of adrenalin but still leaving the vaso-motor apparatus sensitive to camphor. It is reasonable to suppose that drugs with a chloral-like action upon smooth and heart muscle even in doses much smaller than used by Alexander-Lewin might so depress the irritability of the vaso-constrictor muscle as to render small doses of adrenalin ineffective. So it may not be far wrong to attribute the loss of sensitiveness to small doses of adrenalin in cats and rabbits to a loss of irritability in the more advanced stages of chloral or paraldehyde anesthesia instead of to so-called acquired immunity (tolerance). May not Cushny's results with rabbits have been of this nature? Hunt not only observed no loss of irritability toward small doses of adrenalin but secured with rabbits some of his best results.

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[a] The optical activity of this preparation was determined by Mr. Taveau and myself and compares very well with the best measurements that have ever been made, viz, those of Korndörfer (43) made for Flächer. Some of the other readings given in literature are $[\alpha]_D = -51.6^{\circ}$ (Jowett), $[\alpha]_D = -43^{\circ}$ (Pauly), -42.25° (Cushny), $[\alpha]_D^{20^{\circ}} = -50.7^{\circ}$ (Abderhalden and Guggenheim) and $[\alpha]_D^{19.8^{\circ}C} = -51.40^{\circ}$ (Korndörfer) (43).

After trying different anesthetics it was found that ether on the whole was the most satisfactory, and so it was used in all of my later experiments. As is well known the chief objection to this anesthetic is the difficulty of maintaining a uniform condition of anesthesia. But this is practically removed by using a modification of the drop method suggested by Porter. In this way even cats, proverbial for their vasomotor "storms," yielded results that were very consistent. On the whole I prefer morphine along with ether for dogs and ether alone for cats. When working with small doses of adrenalin the best results are assured by an anesthesia just sufficient to maintain a condition of unconsciousness so that the animal has no pain, with only enough curare to render it free from muscle tremors. The amount of curare given must of course be determined by experience, since not only different samples vary but individual animals also vary to a certain extent in their reaction to this drug. Indeed large doses of curare should be avoided. Being a depressant, it lowers the blood pressure and interferes with the action of adrenalin, often making consistent results impossible, even with dogs, which in my experience yield the most reliable results.[a]

In all the later experiments the injections were made from a standardized 1 c. c. syringe graduated in five-hundredths of a c. c. The cannulæ were of small bore, provided with very short connections allowing a minimum amount of dead space. There was one injection set for each of the two solutions to be compared so that one solution might be injected into the right and the other into the left saphenous vein. When constant results were thus obtained the solution formerly used for the left vein was now injected as a check into the right vein from the right injection set and *vice versa*.

The following blood-pressure tables are results selected as typical from 21 animals (cats and dogs). Tables I to VI inclusive represent in a general way the relative physiological activity of synthetic dl-adrenalin and natural l-adrenalin. The latter of these compounds has already been described. The synthetic dl-adrenalin manufactured by Meister, Luscius, and Bruning is said to be a methyl-amino-alcohol or, more specifically, ortho-dioxy-phenyl-ethanol-methyl-amino-hydrochloride represented by the formula

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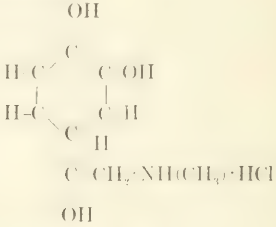
[a] This animal even at the end of a nine-hour experiment yielded with 1 c. c. injections of a 1:100,000 adrenalin solution a rise of blood pressure equivalent to that from the first injections of this amount, showing in no case a diminished sensitiveness to l-natural and dl-synthetic adrenalin unless too much ether was administered.



It is a fine, granular, almost white, crystalline powder, easily soluble in water or normal saline. The solution decomposes much more readily than does a similar solution of the natural l-adrenalin. It turns cherry-red, later brownish, and finally deposits a brown precipitate, whereupon the solution loses its characteristic color and likewise its physiological activity. It is optically inactive and possesses the chemical properties generally attributed to it.

The data of Table I show distinctly that the natural l-adrenalin is more active than the synthetic dl- since equal volumes of 1:100,000 of the two solutions result in rises of blood pressure, which with few exceptions are greater for the natural product than for the synthetic. Although this table shows only a few results with 1:100,000 solutions, a great number of similar injections were made with solutions of them, maintaining the ratio 1 l : 1 dl, but varying in concentration from 1:5,000 to 1:200,000, with the result that the natural base nearly always caused the greatest rise of blood pressure. So that other sets might be chosen to illustrate the same point as does Table I. If instead of the effect of the equal volume of solution of like concentration, a comparison is made of the equal volume of solution with twice as much of the synthetic dl- substance as of the natural l-, the rise of blood pressure from the latter is nearly always less than that resulting from the synthetic dl-, as Table II will show. This table serves to illustrate results with solutions of higher or lower concentration with the ratio 1 l : 2 dl maintained. It is thus evident that with the ratio 1 : 1 the natural l- is too strong to cause like rises of blood pressure, with the ratio 1:2 the natural l- is too weak, and that the right ratio lies between these two. With a gradual increase of the concentration of the weaker or a decrease of the stronger a ratio was found that yielded the greatest number of like rises of blood pressure, which is 2 l:3 dl. Tables III and V, respectively, show that a 1:60,000 solution of natural l- is the equivalent of a 1:40,000 solution of synthetic dl-adrenalin. Table IV shows that like rises of blood pressure may be obtained with equal volumes of 1:30,000 and 1:20,000 solutions of natural l- and synthetic dl-adrenalin, respectively. Table VI illustrates the same thing for cats.

It is also true that isolated cases might be chosen to show that both Biberfeld and Cushny were correct, the former maintaining that these two products are equal in activity, the latter contending that the synthetic dl-product has only one-half the activity of the natural base. For in a large number of experiments one may occasionally obtain equal rises of blood pressure when comparing 1:50,000 of synthetic dl- with 1:100,000 natural l-adrenalin or when comparing solutions of equivalent concentrations. But there is no such agreement after successive sets of readings, as indicated in the accompanying tables that have a ratio of concentration 2:3, instead of 1:1 according to Biberfeld, or 1:2 according to Cushny. Cushny is, however, more nearly correct than Biberfeld, and his experimental data, though inadequate, carries greater weight than any hitherto published. Finally, my results indicate that the ratio of activity of the natural l-base and the synthetic dl-product are to each other as 2:3. These results are in accordance with my first determinations, made before the publication of Cushny's first paper, and I am inclined to think them more nearly correct than his, which made the ratio 1:2.

Table I.—*The relative activity of natural l-adrenalin base and synthetic dl-adrenalin hydrochloride determined by blood pressure.*

Blood-pressure experiment No. 16, January 11, 1909.

Dog, 7,660 gm. weight, subcutaneous injection of 76.6 mg. of morphine sulphate, ether anesthesia, small doses of curare from time to time. Both vagi cut. Artificial respiration of warmed air. Solutions injected into femoral veins. Five milligrams of adrenalin base dissolved in 5 c. c. Ringer 11.50 a. m. Six milligrams synthetic dl-adrenalin hydrochloride crystals dissolved in 5 c. c. Ringer solution 11.55 a. m.

	Injection	Base	Ringer	Time of injection	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C. c.	Gram	C. c.	P. m.	M. m.	M. m.	M. m.
Natural l-adrenalin base..	1	1	100,000	1.22	104	171	67
Synthetic dl-adrenalin..	1	1	100,000	1.24	104	155	51
Natural l-adrenalin.....	1	[illegible]	100,000	1.36	106	170	64
Synthetic dl-adrenalin..	1	[illegible]	100,000	1.33	105	162	57
Natural l-adrenalin.....	1	[illegible]	100,000	[illegible]	97	156	59
Synthetic dl-adrenalin..	1	[illegible]	100,000	[illegible]	[illegible]	134	41
Natural l-adrenalin.....	1	1	100,000	[illegible]	[illegible]	[illegible]	51
Synthetic dl-adrenalin..	1	[illegible]	100,000	[illegible]	106	152	46
Natural l-adrenalin.....	1	[illegible]	100,000	[illegible]	104	147	[illegible]
Synthetic dl-adrenalin..	1	1	100,000	2.31	[illegible]	[illegible]	[illegible]
Natural l-adrenalin.....	1	[illegible]	100,000	[illegible]	104	[illegible]	[illegible]
Synthetic dl-adrenalin..	1	[illegible]	100,000	[illegible]	105	134	[illegible]

From the above data it will be observed that the pure adrenalin is stronger than the synthetic dl-; on the other hand, if one chooses such dilutions as to make the solutions of synthetic twice as concentrated (in terms of base) as the natural adrenalin, then this solution, as shown by the following tables, is the stronger of the two physiologically:

TABLE II. (See legend to Table I.)

	Injection.	Base.	Ringer.	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C. c.	*Gram.*	*C. c.*	*P. m.*	*M. m.*	*M. m.*	*M. m.*
Curare	1½						
Synthetic dl-adrenalin	1	1	50,000	12.56	97	181	84
Natural l-adrenalin	1	1	100,000	12.59	98	162	64
Synthetic dl-adrenalin	1	1	50,000	1.6	93	171	78
Natural l-adrenalin	1	1	100,000	1.3	94	153	59
Synthetic dl-adrenalin	1	1	50,000	1.12	100	180	80
Natural l-adrenalin	1	1	100,000	1.9	102	155	53
Synthetic dl-adrenalin	1	1	50,000	3.5	114	190	76
Natural l-adrenalin	1	1	100,000	3.9	110	180	70
Synthetic dl-adrenalin	1	1	50,000	3.11	114	186	62
Natural l-adrenalin	1	1	100,000	3.15	118	177	59
Curare				3.22			
Synthetic dl-adrenalin	1	1	50,000	3.38	112	187	75
Natural l-adrenalin	1	1	100,000	3.41	115	186	71
Synthetic dl-adrenalin	1	1	50,000	4.7	122	220	98
Natural l-adrenalin	1	1	100,000	4.9	124	214	90
Synthetic dl-adrenalin	1	1	50,000	4.11	122	218	96
Natural l-adrenalin	1	1	100,000	4.15	124	204	80
Synthetic dl-adrenalin	1	1	50,000	4.22	123	194	71
Natural l-adrenalin	1	1	100,000	4.19	122	178	56
Synthetic dl-adrenalin	1	1	50,000	4.28	127	218	91
Natural l-adrenalin	1	1	100,000	4.25	124	210	86
Synthetic dl-adrenalin	1	1	40,000	5.52	95	213	128
Natural l-adrenalin	1	1	80,000	5.49	96	216	120
Synthetic dl-adrenalin	1	1	40,000	6.3	103	197	94
Natural l-adrenalin	1	1	80,000	6.8	104	194	90
Synthetic dl-adrenalin	1	1	40,000	6.13	96	186	90
Natural l-adrenalin	1	1	80,000	6.12	98	168	70

If instead of the concentrations described in Tables I and II the ratio 2 of synthetic to 3 of natural be used, the values representing the rises in blood pressure from 1 c. c. more nearly approximate each other. It is indeed remarkable how different sets of readings agree with each other, how constant is the interval required for recovery of the vasomoter apparatus, and with what certainty one can forecast the height to which the blood pressure will ascend in response to the second injection of a given set. The following tables bring this out more clearly:

TABLE III. (See legend to Table I.)

	Injection.	Base.	Ringer.	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C.c.	Gram.	C.c.	P.m.	M.m.	M.m.	M.m.
Synthetic dl-adrenalin..	1	1	40,000	6.30	93	202	109
Natural l-adrenalin.	1	1	60,000	6.32	94	200	106
Synthetic dl-adrenalin..	1	1	40,000	6.41	84	197	113
Natural l-adrenalin.....	1	1	60,000	6.44	84	197	113
Synthetic dl-adrenalin..	1	1	40,000	6.46	83	194	111
Curare.................	½	....		7.30	.....	...	..
Synthetic dl-adrenalin..	1	1	40,000	7.48	86	198	112
Natural l-adrenalin base.	1	1	60,000	7.50	84	198	114
Synthetic dl-adrenalin..	1	1	40,000	7.54	84	194	110
Natural l-adrenalin.	1	1	60,000	7.56	84	198	114

TABLE IV. (See legend to Table I.)

	Injection.	Base.	Ringer.	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C.c.	Gram.	C.c.	P.m.	M.m.	M.m.	M.m.
Synthetic dl-adrenalin..	1	1	20,000	8.37	64	194	130
Natural l-adrenalin.....	1	1	30,000	8.39	64	190	126
Natural l-adrenalin.....	1	1	30,000	8.43	64	190	126
Synthetic dl-adrenalin..	1	1	20,000	8.45	70	198	128
Natural l-adrenalin....	1	1	30,000	8.47	70	198	128
Synthetic dl-adrenalin..	1	1	20,000	9.00	73	208	135
Natural l-adrenalin.......	1	1	30,000	9.02	73	207	134
Synthetic dl-adrenalin....	1	1	20,000	9.04	72	192	120
Natural l-adrenalin......	1	1	30,000	9.06	72	193	121
Curare......	.	.		9.12	...		
Natural l-adrenalin.......	1	1	30,000	9.18	67	215	148
Synthetic dl-adrenalin..	1	1	20,000	9.20	61	208	147
Natural l-adrenalin.......	1	1	30,000	9.23	64	197	133
Synthetic dl-adrenalin.	1	1	20,000	9.25	61	196	135
Natural l-adrenalin....	1	1	30,000	9.27	62	188	126
Synthetic dl-adrenalin..	1	1	20,000	9.29	60	186	126

TABLE V.

Experiment 17, January 12, 1909.

Pregnant cat, weight 3,770 gm., ether anesthesia, curare. Artificial respiration with warm air. Five milligrams of adrenalin base dissolved in Ringer solution 9 a. m. Six milligrams synthetic dl-adrenalin hydrochloride dissolved in 5 c. c. Ringer solution 10 a. m. Vagi cut 1.12 p. m. Injection into femorals.

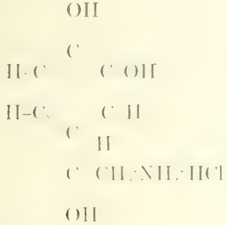
	Injection.	Base.	Ringer.	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C. c.	*Gram.*	*C. c.*	*P. m.*	*M. m.*	*M. m.*	*M. m.*
Natural l-adrenalin .	1	1	90,000	12.18	132	152	20
Synthetic dl-adrenalin.	1	1	60,000	12.19	130	149	19
Natural l-adrenalin....	1	1	90,000	12.21	135	151	16
Synthetic dl-adrenalin.	1	1	60,000	12.23	135	151	16
Natural l-adrenalin....	1	1	90,000	12.38	140	157	17
Synthetic dl-adrenalin..	1	1	60,000	12.41	141	158	17
Natural l-adrenalin....	1	1	90,000	12.49	142	158	16
Synthetic dl-adrenalin.	1	1	60,000	12.57	144	160	16
Vagi cut...............			....	1.12	....	....	....
Curare.................	1½		...	1.21	....	....	....
Natural l-adrenalin...	1	1	60,000	1.32	142	168	26
Synthetic dl-adrenalin.	1	1	40,000	1.34	143	168	25
Natural l-adrenalin.....	1	1	60,000	1.38	142	164	22
Synthetic dl-adrenalin.	1	1	40,000	1.42	148	170	22
Natural l-adrenalin....	1	1	60,000	1.44	146	170	24
Synthetic dl-adrenalin..	1	1	40,000	1.46	149	172	23
Synthetic dl-adrenalin..	1	1	40,000	1.49	146	172	26
Natural l-adrenalin.....	1	1	60,000	1.57	147	172	25

TABLE VI. (See legend to Table V.)

	Injection.	Base.	Ringer.	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C. c.	*Gram.*	*C. c.*	*P. m.*	*M. m.*	*M. m.*	*M. m.*
Natural l-adrenalin..	1	1	30,000	3.12	122	156	34
Synthetic dl-adrenalin	1	1	20,000	3.15	122	153	31
Synthetic dl-adrenalin..	1	1	20,000	3.29	124	148	24
Natural l-adrenalin .	1	1	30,000	3.31	120	147	25
Synthetic dl-adrenalin.. . .	1	1	20,000	3.33	123	149	26

THE RELATIVE ACTIVITY OF ORTHO-DIOXY-PHENYL-ETHANOL-AMIN (ARTERENOL) AND NATURAL L-ADRENALIN DETERMINED BY BLOOD PRESSURE.

Another synthetic product of interest is the reduction product of amino-aceto-pyro-catechin, dioxy-phenyl-ethanol-amin. The chloride is known commercially as arterenol hydrocholride, the formula of which is given as—



It is a fine, granular, odorless, crystalline powder, easily soluble in water or normal saline. The deterioration of this product is not very evident, since there does not accompany it distinct coloration so easily noticed in adrenalin solutions. Just how rapid this process of deterioration is has not been determined, but it certainly is an important factor in determining the relative value of the substance. One solution received for purposes of testing was found to have an activity comparable to that of our natural l-adrenalin. So surprised was I to note this that the balance of the sample was preserved for a subsequent testing, but a few days later this preparation had deteriorated and a fresh solution had to be made. It can readily be seen that to the physician the commercial 1:1,000 solution may prove very disappointing if kept for subsequent use after once the original package has been opened. The fresh solution, however, has a remarkable vaso-constrictor action, and if it were more certain in yielding quantitative results throughout entire experiments, it would be a worthy rival of natural l-adrenalin itself. In the early parts of long experiments fairly constant results may be obtained with small doses, but in the latter parts, or after larger doses (1 c. c. of 1:60,000 or over), irregularities seemed to appear the exact meaning of which must be determined later. As a matter of fact, the following tables represent results taken from the beginning of experiments only.

Table VIII is interesting, since it shows that a 1 c. c. injection of adrenalin or arterenol in 1:100,000 solutions causes a like rise of blood pressure (27-28 m. m.) and for 1 c. c. injections of a 1:50,000 solution (38 m. m.). A 1:50,000 solution of arterenol, however, is uniformly more active than a 1:100,000 solution of natural l-base. Finally, consistent results are obtained by comparing the two substances each in a concentration of 1:80,000 or in one of 1:60,000.

TABLE VII. *Physiological activity of arterenol hydrochloride compared with that of l-natural adrenalin base.*

Blood pressure experiment No. 21, March 22, 1909.

Female bull terrier weight 10.1 kilograms; 10.20 a. m. subcutaneous injection of 100 milligrams morphine sulphate. Ether anesthesia; small intravenous injections of curare from time to time. Both vagi tied off. Artificial respiration with warmed air. Solutions injected into saphenous veins. 5 milligrams specially pure natural l-adrenalin base dissolved (10.35 a. m.) in 5 c. c. of Ringer solution acidulated with calculated amount of HCl. Six milligrams arterenol hydrochloride dissolved in 5 c. c. Ringer solution 11.50 a. m. These two solutions were then diluted as if they were the equivalents of a 1:1,000 solution of the base—that is, 1 c. c. of either one of these stock solutions diluted to 10 c. c. gave a 1:10,000 solution (base).

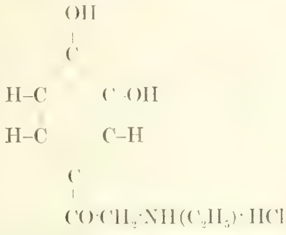
	Injection	Base.	Ringer.	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C. c.	*Gram.*	*C. c.*	*P. m.*	*M. m.*	*M. m.*	*M. m.*
Natural l-adrenalin base..	1	1	100,000	12.05	127	182	55
Arterenol hydrochloride..	1	1	100,000	12.09	126	182	56
Natural l-adrenalin base...	1	1	100,000	12.14	127	184	57
Arterenol hydrochloride...	1	1	100,000	12.18	124	179	55
Curare................			....	12.21	....	....	.....
Natural l-adrenalin base...	1	1	100,000	12.28	130	193	63
Arterenol hydrochloride..	1	1	100,000	12.31	121	184	63
Arterenol hydrochloride...	1	1	100,000	1.08	131	152	21
Natural l-adrenalin base..	1	1	100,000	1.12	131	150	19
Arterenol hydrochloride..	1	1	100,000	1.16	134	156	22
Natural l-adrenalin base..	1	1	100,000	1.20	134	155	21
Arterenol hydrochloride....	1	1	100,000	1.28	132	154	22
Curare	2		....	1.44	....	....	....
Arterenol hydrochloride...	1	1	100,000	2.47	142	185	43
Natural l-adrenalin base..	1	1	100,000	2.50	151	195	44

TABLE VIII. (See legend to Table V.)

	Injection.	Base.	Ringer.	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C. c.	*Gram.*	*C. c.*	*P. m.*	*M. m.*	*M. m.*	*M. m.*
Natural l-adrenalin base..	1	1	80,000	6.16	157	187	30
Arterenol hydrochloride..	1	1	80,000	6.20	153	184	31
Natural l-adrenalin base..	1	1	100,000	7.24	123	150	27
Arterenol hydrochloride..	1	1	50,000	7.26	124	162	38
Natural l-adrenalin base..	1	1	100,000	7.30	126	154	28
Arterenol hydrochloride..	1	1	50,000	7.35	126	164	38
Arterenol hydrochloride..	1	1	60,000	7.39	124	152	28
Natural l-adrenalin base..	1	1	60,000	7.42	128	155	27

THE RELATIVE ACTIVITY OF ETHYL-AMINO-ACETO-CATECHOL AND NATURAL L-ADRENALIN AS DETERMINED BY BLOOD PRESSURE.

A substance known commercially as homorenon hydrochloride was the last one tested. This is the hydrochloride of ethyl-amino-aceto-catechol represented by the formula:



It is a white powder composed of fine crystalline needles, easily soluble in normal saline, and, so far as I have been able to determine, keeps much better than any of the products already mentioned. Its optical activity is *nil.* As a vaso-constrictor it is inferior to all of the other substances tested, as illustrated in Tables IX and X. A 1 c. c. injection of a 1:160,000 solution of natural l-adrenalin causes a rise of blood pressure which with a like volume of homorenon is equaled only by a 1:2,000 solution. It is found that a 1 c. c. injection of a 1:80,000 solution of adrenalin base causes the same rise of blood pressure as a 1 c. c. injection of a 1:1,000 solution of homorenon. So that by the blood-pressure method, natural l-adrenalin base is eighty times as active as homorenon base the base being calculated from the foregoing formula.

TABLE IX.—*Physiological activity of homorenon hydrochloride compared with that of l-natural adrenalin base.*

Blood-pressure experiment No. 20. March 16, 1909.

Female fox terrier, weight 6.8 kilograms. 10.15 a. m. subcutaneous injection of 68 mg. of morphine sulphate. Ether anesthesia, small intravenous injections of curare from time to time. Both vagi tied off. Artificial respiration of warmed air. Solutions injected into saphenous veins.

Five milligrams specially pure adrenalin l-base dissolved 10.23 a. m. in 5 c. c. of Ringer solution, acidulated with the calculated amount of HCl.

Six milligrams arterenol hydrochloride dissolved 6 p. m. in 5 c. c. Ringer solution.

118.6 mg. homorenon hydrochloride dissolved 11.30 a. m. in 10 c. c. of Ringer solution. These solutions were then diluted as if they were the equivalents of a 1:1,000 solution of adrenalin base.

	Injection	Base	Ringer	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C. c.	Grm.	C. c.	P. m.	M. m.	M. m.	M. m.
Natural l-adrenalin base.	1	[illegible]	160,000	3.49	[illegible]	204	[illegible]
Homorenon hydrochloride............	1	[illegible]	2,000	[illegible]	178	202	[illegible]
Natural l-adrenalin base..............	1	1	160,000	[illegible]	[illegible]	202	[illegible]
Natural l-adrenalin base..............	1	1	160,000	[illegible]	160	[illegible]	[illegible]
Homorenon hydrochloride............	1	1	2,000	[illegible]	150	175	[illegible]

TABLE IX.—*Physiological activity of homorenon hydrochloride compared with that of l-natural adrenalin base*—Continued.

	Injection.	Base.	Ringer.	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C. c.	*Gram.*	*C. c.*	*P. m.*	*M. m.*	*M. m.*	*M. m.*
Natural l-adrenalin base...	1	1	160,000	4.31	156	178	22
Homorenon hydrochloride.	1	1	2,000	4.36	154	177	23
Natural l-adrenalin base...	1	1	160,000	5.15	172	186	21
Homorenon hydrochloride.	1	1	2,000	5.19	173	195	22
Natural l-adrenalin base..	1	1	160,000	5.22	172	194	22
Homorenon hydrochloride..	1	1	2,000	5.25	[illegible]	191	22
Homorenon hydrochloride	1	1	2,000	5.32	[illegible]	176	21

TABLE X. (See legend to VI.)

	Injection.	Base.	Ringer.	Time of injection.	Blood pressure before.	Blood pressure after.	Rise of blood pressure.
	C. c.	*Gram.*	*C. c.*	*P. m.*	*M. m.*	*M. m.*	*M. m.*
Natural l-adrenalin base..	1	1	80,000	1.57	138	180	42
Homorenon hydrochloride.	1	1	1,000	2.1	138	180	42
Natural l-adrenalin base	1	1	80,000	2.28	158	208	50
Homorenon hydrochloride.	1	1	1,000	2.33	156	205	49
Natural l-adrenalin base..	1	1	80,000	2.39	166	215	49
Homorenon hydrochloride	1	1	1,000	2.56	164	200	36
Natural adrenalin base .	1	1	80,000	3.02	162	198	36
Homorenon hydrochloride	1	1	1,000	3.04	156	193	37
Natural adrenalin base .	1	1	80,000	3.09	160	198	38
Natural adrenalin base	1	1	80,000	3.39	174	228	54
Homorenon hydrochloride.	1	1	1,000		170	223	53

THE RELATIVE TOXICITY OF ORTHO-DIOXY-PHENYL-ETHANOL-METHYL-AMIN (NATURAL L- AND SYNTHETIC DL-ADRENALIN), OF ORTHO-DIOXY-PHENYL-ETHANOL-AMIN ("ARTERENOL"), AND ETHYL-AMINO-DIOXY-ACETO-PHENON ("HOMORENON") AS DETERMINED UPON MICE.

It is conceivable that coefficients of physiological activity of a series of closely related compounds, when determined by vasomotor effects, may be so arranged as to express one ratio, whereas those determined by toxic effects when similarly arranged may express a very different one. That is, a coefficient determined by the blood-pressure method may be relatively small when compared with that determined by the dose necessary to kill, say, more than 50 per cent of the animals injected. It would seem that a comparison of such coefficients determined in various ways might throw additional light on the relative safety and effectiveness of adrenalin-like bodies. But scarcely anything has been published from which can be derived the coefficients needed in making the above-named comparisons. Hence, with a view of determining the relative toxicity of the compounds already compared by the blood-pressure method, the following experiments were undertaken. The test object chosen is the ordinary white mouse, reared in our own mousery under conditions well under control. As a rule these mice when placed in individual jars on a diet of oats and water lose in weight, but after the first day, upon becoming accustomed to their new surroundings, return to normal. Generally on a given day the mice were brought into the preparation room, weighed, and on the second day weighed again, whereupon the dose was calculated and unless otherwise stated the solution made up and injected at once. All mice were injected tailward underneath the skin of the back. The syringe used was calibrated in 0.05 c. c., standardized, and fitted at first with a steel needle. Since this needle, especially if tarnished, decomposed the solutions, a platinum-iridium needle was substituted.

The phenomena accompanying subcutaneous injections of this nature may be summarized as follows: Soon after being released the animal seeks a corner of the jar, under the chaff, or at once stretches itself lengthwise upon the belly with hind feet directed backward and planta turned upward. The favorite position is to lie upon the cool, moist oat jar with head downward, breathing at first very rapidly and later more or less irregularly. In this stage the animal may be highly excitable, its reflexes being probably intensified not only by the injected material but also by the fear aroused by the smarting of the needle's prick and by fright from handling. In case the dose is rather large the bulbus is protruded, and the glands of the eye are usually stimulated to excessive secretion.

In some mice thirty to sixty mintues after injection a small opalescent [a] disc appears in the eye. This later becomes opaque white and may be confined to only one or extended to both eyes. The phenomenon seems to be one of the lens and resembles cataract. In this respect it differs from the opaquing observed in the eyes of frogs, for in the latter case I have proved that the initial opaquing is due to coagulation of the outer membranes of the bulbus and that it can be removed by stripping off this membrane, whereupon the eye becomes perfectly clear. In the eye of the mouse the opaque disc, examined with a magnifying glass, seems to be underneath the cornea, of the size and position of the lens. The following protocol is taken from a mouse injected with arterenol and is typical of the cases observed with natural l- and synthetic dl-adrenalin:

Mouse 324, weight 22.22 gm. March 15, 12.20 p. m., injected subcutaneously 0.44 c. c. (0.88 mg.) arterenol hydrochloride.

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[a] After this section had been written I noticed in a recent article by [illegible] a reference to his observation of what seems to be a similar phenomenon.

12.26, lies flat, restless from time to time, hind leg stretched out, but when touched is jerked away.

12.30, forced breathing.

12.33, abundant salivation, leg still sensitive, but control of hip muscle imperfect.

12.37, lies flat; does not move when tail is pinched; feet and leg still sensitive, but reflex not so rapid as before.

1.34, opaque disc in center of left eye.

2.35, opaque disc of left eye much larger, opaque disc in right eye also.

March 16, 8.15 a. m. Both eyes cleared up, mouse prostrated.

March 17, 9 a. m. Mouse prostrated, eye clear, breathing shallow and slow.

12.20, found dead.

The saliva may or may not flow abundantly, but when it does the amount is so great as to drop from the lower jaw. In the males erection may follow and even be accompanied by semination. Should the dose be lethal the earlier stages are passed through quickly; in general the animal becomes very sluggish, largely on account of constriction of the blood vessels, causing anaemia not only of the muscles, but also of the cord and brain. Sometimes animals die very suddenly from doses that seem hardly to affect other individuals. I am inclined to think that in such cases some of the drug enters a vein. Otherwise there does not occur any explanation, unless it be due to heart failure, such as indeed has been noted in apparently vigorous human beings under the influence of great fear or excitement. When spasms result, the animal usually dies in one to two minutes; only one out of over four hundred recovered, and this one died at the end of four or five days. When once the lethal dose is exceeded death becomes more violent, accompanied by spasms of very short duration.

TOXICITY OF NATURAL L-ADRENALIN.

Judging from the remarkable activity of the natural l-adrenalin in causing vaso-constriction, it was presumed that the lethal subcutaneous dose would be small. The experimental data of Table I justifies this presumption, for 0.008 mg. per gram mouse is usually fatal. Certain mice may die from doses as small as 0.004 mg. per gram, but this is the exception, whereas others may survive as much as 0.017 mg. per gram, which is likewise unusual. Perhaps a greater number of experiments might reveal certain cases of even greater resistance, but in spite of these exceptions, as seen in Table XI, it may be said that 0.008 is the lethal dose for the average mouse.

TABLE XI.—*Toxicity of pure natural l-adrenalin.*

Number of mouse.[a]	Mg. base per gm.[b]	Body weight.	Injected.			Died.
			Total.		Time.	
		Grams.	*Mg.*	*C. c.*		
42	0.0005	16.45	0.008	0.04	Dec. 3, 9.28 a. m.	
43	0.00075	19.28	0.014	0.07	Dec. 3, 9.32 a. m.	
44	0.001	16.62	0.017	0.08	Dec. 3, [illegible] a. m.	
52	0.001	20.92	0.021	0.10	Dec. 4, 9.28 a. m.	
32	0.002	18.23	0.036	0.07	Dec. 2, 9.29 a. m.	
45	0.002	20.92	0.042	0.22	Dec. 3, 9.37 a. m.	
53	0.002	21.21	0.042	0.21	Dec. 4, 9.26 a. m.	
62	0.002	12.20	0.024	0.02	Dec. 5, 9.08 a. m.	
72	0.002	16.52	0.033	0.03	Dec. 7, 9.40 a. m.	
82	0.002	20.40	0.040	0.04	Dec. 7, 2.01 p. m.	
22	0.004	18.52	0.074	0.08	Nov. 30, 4.34 p. m.	Nov. 30, 4.48 p. m.
33	0.004	17.89	0.071	0.15	Dec. 2, 9.34 a. m.	Dec. 2, 11.35 a. m.
46	0.004	19.77	0.079	0.42	Dec. 3, 9.39 a. m.	
54	0.004	17.33	0.069	0.35	Dec. 4, 9.28 a. m.	
63	0.004	15.80	0.063	0.063	Dec. 5, 9.11 a. m.	
73	0.004	17.74	0.071	0.07	Dec. 7, 9.43 a. m.	
83	0.004	20.00	0.080	0.08	Dec. 7, 2.05 p. m.	
55	0.005	16.14	0.081	0.41	Dec. 4, 9.32 a. m.	
56	0.006	13.62	0.082	0.41	Dec. 4, 9.34 a. m.	
64	0.006	20.05	0.120	0.12	Dec. 5, 9.13 a. m.	
74	0.006	14.54	0.087	0.09	Dec. 7, 9.45 a. m.	
84	0.006	17.28	0.103	0.10	Dec. 7, 2.07 p. m.	
112	0.006	12.41	0.074	0.07	Dec. 9, 5.21 p. m.	
152	0.006	17.77	0.107	0.11	Jan. 4, 2.52 p. m.	
155	0.006	16.55	0.099	0.10	Jan. 4, 2.55 p. m.	Jan. 5, 12.15 a. m. (W.)
158	0.006	13.07	0.078	0.08	Jan. 4, 3.08 p. m.	
23	0.008	18.15	0.145	0.16	Nov. 30, 4.37 p. m.	Nov. 30, 4.43 p. m.
34	0.008	15.98	0.128	0.14	Dec. 2, [illegible] a. m.	Dec. 2, 12.34 p. m.
65	0.008	20.51	0.164	0.16	Dec. 5, 9.16 a. m.	Dec. 5, 10 p. m.
75	0.008	20.50	0.164	0.16	Dec. 7, 9.48 a. m.	Dec. 7, 6 to 7 p. m.
85	0.008	16.00	0.128	0.13	Dec. 7, 2.10 p. m.	
113	0.008	16.30	0.130	0.13	Dec. 9, 5.23 p. m.	Dec. 9, 8 p. m. (W.)
153	0.008	18.27	0.146	0.15	Jan. 4, 2.53 p. m.	
156	0.008	16.65	0.133	0.13	Jan. 4, [illegible] p. m.	
159	0.008	13.79	0.110	0.11	Jan. 4, 3.10 p. m.	
218	0.008	19.39	0.160	0.16	Jan. 9, 3.07 p. m.	Jan. 9, 3.20 p. m.
221	0.008	15.12	0.120	0.12	Jan. 9, 3.13 p. m.	Jan. 10, 10.30 a. m. (W.)
224	0.008	13.61	0.110	0.11	Jan. 9, 4.51 p. m.	Jan. 10, [illegible] (W.)
227	0.008	13.78	0.110	0.11	Jan. 9, 5.01 p. m.	Jan. 10, 10.30 a. m. (W.)
230	0.008	14.84	0.120	0.12	Jan. 9, 5.10 p. m.	Jan. 10, 3.30 a. m. (W.)
66	0.010	12.66	0.127	0.13	Dec. 5, 9.19 a. m.	Dec. 5, 4.20 p. m.
76	0.010	17.24	0.170	0.17	Dec. 7, 9.50 a. m.	Jan. 7, 2.26 p. m.
86	0.010	13.04	0.130	0.13	Dec. 7, 2.12 p. m.	
114	0.010	20.28	0.203	0.20	Dec. 9, 5.25 p. m.	
154	0.010	19.04	0.190	0.19	Jan. 4, 2.55 p. m.	[illegible]
157	0.010	16.51	0.165	0.16	Jan. 4, 3.05 p. m.	[illegible] (W.)
160	0.010	13.00	0.130	0.13	Jan. 4, [illegible] p. m.	
215	0.010	14.55	0.150	0.15	Jan. 9, 3.00 p. m.	Jan. 10, 12.55 p. m. (W.)
216	0.010	18.04	0.180	0.18	Jan. 9, 3.03 p. m.	Jan. 9, 3.22 p. m.

[a] Mice not marked "W" or "F" were observed by the author [illegible]

[b] These figures are only approximate. The actual doses [illegible]

W. Time noted by watchman.

TABLE XI.—*Toxicity of pure natural l-adrenalin*—Continued.

Number of mouse.	Mg. base per gm.	Body weight.	Injected.			Died.
			Total.		Time.	
		Grams	*Mg.*	*C. c.*		
219	0.010	12.73	0.130	0.13	Jan. 9, 3.00 p. m.	
222	0.010	13.58	0.140	0.14	Jan. 9, 3.15 p. m.	
225	0.010	15.44	0.150	0.15	Jan. 9, 4.57 p. m.	Jan. 10, 3.30 a. m. (W.)
228	0.010	13.72	0.140	0.14	Jan. 9, 5.05 p. m.	Jan. 10, 5.14 p. m.
231	0.010	15.77	0.160	0.16	Jan. 9, 5.12 p. m.	Jan. 9, 5.28 p. m.
115	0.012	13.74	0.165	0.16	Dec. 9, 5.27 p. m.	Dec. 9, 6.30 p. m. (W.)
217	0.012	14.80	0.180	0.18	Jan. 9, 3.05 p. m.	
220	0.012	19.83	0.240	0.24	Jan. 9, 3.12 p. m.	Jan. 12, 8 a. m. (W.)
223	0.012	17.80	0.210	0.21	Jan. 9, 3.19 p. m.	Jan. 9, 3.35 p. m.
226	0.012	13.04	0.140	0.14	Jan. 9, 4.59 p. m.	Jan. 9, 5.11 p. m.
229	0.012	12.11	0.140	0.14	Jan. 9, 5.07 p. m.	Jan. 9, 5.26 p. m.
232	0.012	13.22	0.140	0.14	Jan. 9, 5.15 p. m.	Jan. 9, 5.22 p. m.
24.	0.013	15.77	0.210	0.21	Nov. 30, 4.40 p. m.	Dec. 1, 9 p. m. (F.)
35.	0.013	13.43	0.180	0.18	Dec. 2, 9.21 a. m.	Dec. 2, 11 a. m.
116	0.014	16.78	0.230	0.23	Dec. 9, 5.29 p. m.	Dec. 10, 12.15 a. m.
12	0.0147	18.27	0.270	0.27	Nov. 28	Nov. 28, 4.40 p. m. (W.)
7	0.017	18.20	0.318	0.32	Nov. 23, 1.18 p. m.	
8	0.017	17.09	0.298	0.30	Nov. 23	Nov. 25, 10 p. m.
13	0.017	20.18	0.350	0.35	Nov. 28, 3.18 p. m.	Nov. 28, 3.29 p. m.
25.	0.017	17.94	0.300	0.30	Nov. 30, 4.43 p. m.	Nov. 30, 4.55 p. m.
26.	0.0218	16.75	0.370	0.37	Nov. 30, 4.47 p. m.	Dec. 1, 1.45 a. m. (W.)
14	0.0218	21.52	0.470	0.47	Nov. 28, 3.22 p. m.	Nov. 28, 6.30 p. m. (W.)
36.	0.022	17.97	0.410	0.41	Dec. 2, 9.23 a. m.	Dec. 2, 9.28 a. m.
9	0.026	12.38	0.320	0.32	Nov. 23, 10.54 a. m.	Nov. 27, 1.50 a. m. (F.)
10.	0.035	14.40	0.500	0.50	Nov. 25, 11.23 p. m.	Nov. 26, 6 p. m. (F.)
15.	0.035	19.26	0.590	0.59	Nov. 28, p. m.	Nov. 30, 8.55 p. m. (F.)

F. Found dead at time noted.
W. Time noted by watchman.

In order to avoid errors from deterioration the solutions were usually made up just before using, 5 mgs. of the base having been weighed up and dissolved in 5 c. c. of Ringer solution. The solvent was acidified with hydrochloric acid slightly in excess of the amount necessary to transform the adrenalin into the chloride. If more specific information is desired as to the age of a solution at the time of any given injection it is easily obtained by the aid of the following data in conjunction with the time of the injection given in the tables:

Mice Nos. 12, 13, 14, 15, 16, solution made up about 9 a. m. November 27, 1908.

Mice Nos. 42 to 46, inclusive, solution made up about 11 a. m. December 2, 1908.

Mice Nos. 52 to 56, inclusive, solution made up about 9 a. m. December 4, 1908, and diluted to 1:5,000.

Mice Nos. 62 to 66, inclusive, the solution was made up between 9 and 11 a. m. December 4, 1908.

Mice Nos. 72 to 76, inclusive, the solution was made up 11.50 a. m. December 5, 1908.

Mice Nos. 72, 73, 74, 75, and 76 had been injected with 0.008, 0.014, 0.017, 0.044, and 0.085 mg. of adrenalin, respectively.

Mice Nos. 82, 83, 84, 85, and 86, solution made up 11.50 a. m. December 5. These mice had been injected on December 4 with 0.021, 0.042, 0.069, 0.081, and 0.082 mg. of adrenalin, respectively.

Nos. 112 to 116, inclusive, solution made up 11.30 a. m. December 9.

Nos. 152 to 160, inclusive, solution made up about 2.30 p. m. January 4, 1909.

Nos. 215 to 232, inclusive, solution made up between 2 and 3 p. m. January 9.

TOXICITY OF SYNTHETIC DL-ADRENALIN.

If instead of natural l-adrenalin synthetic dl-adrenalin hydrochloride be used the symptoms of toxicity are practically the same as those described for adrenalin. There is, however, greater irregularity in the results. In exceptional cases 0.006 to 0.008 mg. per gram mouse may cause death. (Table XII.) By increasing the size of the initial dose the number of deaths likewise becomes larger, so that injections of 0.012 mg. may result in a death rate even as high as does one of 0.020 mg. per gram body weight. The lethal dose, therefore, may be said to be from 0.012 to 0.016 mg. per gram. This would make the natural product 1.5 to 2 times as toxic as the synthetic. It is interesting to observe that the toxicity of the two substances is in direct proportion to their vaso-constrictor action as measured by the blood-pressure method.

TABLE XII.—*Toxicity of synthetic dl-adrenalin hydrochloride.*

Number of mouse.[a]	Mg. base per gm[b]	Body weight.	Injected.			Died.
			Total.		Time.	
		Grams.	*Mg.*	*C. c.*		
102	0.002	15.48	0.030	0.03	Dec. 9, 9.45 a. m.	
142	0.002	26.57	0.053	0.05	Dec. 10, 3.43 p. m.	
147	0.002	25.27	0.05	0.05	Dec. 10, 3.56 p. m.	
103	0.004	14.44	0.06	0.06	Dec. 9, 9.48 a. m.	
143	0.004	23.24	0.092	0.09	Dec. 10, 3.45 p. m.	
148	0.004	17.43	0.069	0.07	Dec. 10, 3.58 p. m.	
104	0.006	15.22	0.091	0.09	Dec. 9, 9.51 a. m.	
144	0.006	23.50	0.141	0.14	Dec. 10, 3.48 p. m.	
149	0.006	23.00	0.138	0.14	Dec. 10, 3.59 p. m.	
343	0.006	13.56	0.081	0.08	Mar. 15, 3.10 p. m.	Mar. 15, 3.32 p. m.
344	0.006	11.77	0.070	0.07	Mar. 15, 3.11 p. m.	
345	0.006	12.97	0.077	0.07	Mar. 15, 3.12 p. m.	
346	0.006	13.34	0.080	0.08	Mar. 15, 3.14 p. m.	
347	0.006	14.72	0.088	0.08	Mar. 15, 3.16 p. m.	
105	0.008	17.24	0.138	0.14	Dec. 9, 9.53 a. m.	Dec. 10, 12 m.
145	0.008	24.21	0.194	0.19	Dec. 10, 3.50 p. m.	
150	0.008	24.24	0.194	0.19	Dec. 10, 4.01 p. m.	
348	0.008	20.46	0.163	0.16	Mar. 15, 3.18 p. m.	
349	0.008	16.15	0.130	0.13	Mar. 15, 3.20 p. m.	Mar. 15, 5.30 p. m. (F.).
350	0.008	15.39	0.123	0.12	Mar. 15, 3.22 p. m.	
351	0.008	13.84	0.110	0.11	Mar. 15, 3.24 p. m.	
106	0.010	15.87	0.159	0.16	Dec. 9, 9.56 a. m.	Dec. 10, 12.15 a. m. (W.).
146	0.010	22.46	0.225	0.22	Dec. 10, 3.53 p. m.	
151	0.010	19.37	0.193	0.19	Dec. 10, 4.03 p. m.	
353	0.010	15.45	0.154	0.15	Mar. 15, 3.34 p. m.	
354	0.010	11.80	0.118	0.12	Mar. 15, 3.35 p. m.	
355	0.010	17.38	0.173	0.17	Mar. 15, 3.37 p. m.	Mar. 16, 8 a. m. (F.).

[a] Mice not marked "W" or "F" were observed by the author to die at the time noted.

[b] These figures are only approximate. The actual dose is in column "Total milligrams injected."

W. Time noted by watchman.

F. Found dead at time noted.

Table XII.—*Toxicity of synthetic dl-adrenalin hydrochloride*—Continued.

Number of mouse.	Mg. base per gm.	Body weight	Injected.			Died.
			Total		Time	
		Grams	*Mg.*	*C. c.*		
356	0.010	12.26	0.122	0.12	Mar. 15, 3.39 p. m.	
357	0.010	18.47	0.184	0.18	Mar. 15, 3.40 p. m.	Mar. 17, 1.15 a. m.
161	0.012	18.09	0.217	0.22	Jan. 4, 3.25 p. m.	
164	0.012	15.11	0.181	0.18	Jan. 4, 3.35 p. m.	Jan. 6, 2.30 p. m.
167	0.012	15.18	0.182	0.18	Jan. 4, 3.39 p. m.	Jan. 5, 12.15 a. m. (W.).
188	0.012	14.55	0.174	0.17	Jan. 8, 11.29 a. m.	
191	0.012	16.20	0.194	0.19	Jan. 8, 11.44 a. m.	Jan. 9, 1.30 a. m. (W.).
194	0.012	15.15	0.181	0.18	Jan. 8, 11.52 a. m.	Jan. 10, 10.30 a. m. (W.).
358	0.012	15.00	0.180	0.18	Mar. 15, 3.42 p. m.	
359	0.012	13.62	0.136	0.13	Mar. 15, 3.44 p. m.	
360	0.012	13.49	0.135	0.13	Mar. 15, 3.46 p. m.	
361	0.012	15.80	0.158	0.15	Mar. 15, 3.47 p. m.	
362	0.012	20.24	0.202	0.20	Mar. 15, 3.48 p. m.	
162	0.016	19.91	0.319	0.32	Jan. 4, 3.28 p. m.	Jan. 5, 1.30 a. m. (W.).
165	0.016	15.92	0.255	0.25	Jan. 4, 3.36 p. m.	Jan. 6, 10.05 a. m.
168	0.016	15.35	0.246	0.25	Jan. 4, 3.44 p. m.	
189	0.016	14.35	0.230	0.23	Jan. 8, 11.40 a. m.	Jan. 10, 10.30 a. m. (W.).
192	0.016	21.54	0.345	0.34	Jan. 8, 11.48 a. m.	Jan. 8, 4.50 p. m. (F.).
195	0.016	14.08	0.225	0.23	Jan. 8, 12.05 p. m.	Jan. 10, 10.30 a. m. (W.).
163	0.020	21.37	0.427	0.43	Jan. 4, 3.32 p. m.	
166	0.020	17.85	0.357	0.36	Jan. 4, 3.38 p. m.	Jan. 5, 12.15 a. m. (W.).
169	0.020	15.54	0.311	0.31	Jan. 4, 3.47 p. m.	
190	0.020	17.53	0.350	0.35	Jan. 8, 11.42 a. m.	Jan. 8, 4.50 p. m. (F.).
193	0.020	19.30	0.386	0.39	Jan. 8, 11.50 a. m.	Jan. 9, 2.30 a. m. (W.).
196	0.020	12.54	0.250	0.25	Jan. 8, 12.07 p. m.	

W. Time noted by watchman.
F. Found dead at time noted.

Note.—Concentration of the solutions made from synthetic dl-adrenalin hydrochloride crystals and th time of mixing them are as follows:

Nos. 102 to 106, inclusive, 6 mg. crystals in 5 c. c. Ringer solution about 9 a. m. December 9, 1908.
Nos. 142 to 151, inclusive, 6 mg. crystals dissolved in 5 c. c. of Ringer solution just before using.
Nos. 161 to 169, inclusive, 6 mg. crystals dissolved in 5 c. c. of Ringer solution about 3 p. m. January 4, 190

TOXICITY OF ARTERENOL HYDROCHLORIDE.

Arterenol (see Table XIII) differs from natural l- and synthetic dl products in two essentials; (1) it is far less toxic, and (2) it is, as sai before, more variable in its action. If the animal can survive th first tremendous strain upon the organism, it has greater chances o recovery from arterenol than from doses of adrenalin causing a cor responding degree of prostration. If the animal is again injecte after recovery from sublethal doses, the resulting prostration is as rule correspondingly less severe, and doses that are usually lethal n longer proved to be so. This is illustrated by mice in groups (a) and (b (Table XIII). In the one case 0.040 mg. and in the other 0.080 mg. pe gram mouse is with one exception in each group successfully resiste Whether or not this is a case of acquired tolerance or whether it is mere accident that these groups are made up of unusually resistant mice, is difficult to say. A greater number of experiments will have to be made to decide positively. Nevertheless, the blank spaces in the last column of the table indicate a resistance to the second dose and is very suggestive of the idea that the first injections are a determining factor in making this resistance possible.

At any rate, it is highly important in experiments of this nature that only the results obtained with fresh mice be compared with each other. When this is done it will be observed (Table XIII) that as small a dose as 0.008 mg. per gram body weight may cause death; on the other hand, even three times this dose kills only 1 mouse out of 14. The dose must be 0.040 mg. per gram mouse before any appreciable number die, though not a few mice may survive a dose as large as 0.080 mg. per gram. It is then safe to say that the lethal dose for the average mouse is about 0.040 mg. per gram body weight. If the lethal dose be placed at 0.040 mg. per gram, then natural l-adrenalin is five times as toxic as arterenol.

Table XIII.—*Toxicity of arterenol hydrochloride.*

Number of mouse.[a]	Mg. base per gm.[b]	Body weight.	Injected.			Died.
			Total.		Time.	
		Grams.	Mg.	C. c.		
67	0.004	14.24	0.057	0.06	Dec. 5, 9.23 a. m.	
107	0.004	15.43	0.061	0.06	Dec. 9, 9.22 a. m.	
68	0.008	16.50	0.132	0.13	Dec. 5, 9.28 a. m.	
108	0.008	18.34	0.146	0.15	Dec. 9, 9.24 a. m.	
69	0.012	19.19	0.240	0.24	Dec. 5, 9.30 a. m.	Dec. 8, 12.45 p. m.
109	0.012	17.26	0.207	0.21	Dec. 9, 9.27 a. m.	
117	0.012	16.23	0.195	0.20	Dec. 9, 5.36 p. m.	
179	0.012	14.47	0.173	0.17	Jan. 5	
182	0.012	19.77	0.237	0.24	Jan. 5	
185	0.012	16.87	0.200	0.20	Jan. 5	
70	0.016	17.20	0.275	0.27	Dec. 5, 9.34 a. m.	
110	0.016	19.94	0.318	0.32	Dec. 9, 9.30 a. m.	Dec. 11, 9.10 a. m. (F.).
118	0.016	17.18	0.275	0.27	Dec. 9, 5.38 p. m.	Dec. 9, 5.43 p. m.
180	0.016	18.77	0.300	0.30	Jan. 5	
183	0.016	18.90	0.302	0.30	Jan. 5	
186	0.016	14.54	0.233	0.23	Jan. 5	
111	0.020	10.56	0.211	0.21	Dec. 9, 9.37 a. m.	
119	0.020	14.80	0.296	0.30	Dec. 9, 5.40 p. m.	
181	0.020	16.30	0.326	0.33	Jan. 5	
184	0.020	15.92	0.318	0.32	Jan. 5	
187	0.020	15.92	0.318	0.32	Jan. 5	
120	0.024	19.13	0.459	0.46	Dec. 9, 5.43 p. m.	Dec. 12.
197	0.024	15.63	0.370	0.37	Jan. 8, 1.25 p. m.	
200	0.024	13.62	0.330	0.33	Jan. 8, 1.30 p. m.	
203	0.024	13.32	0.320	0.32	Jan. 8, 1.35 p. m.	

[a] Mice not marked "W" or "F" were observed by the author to die at the time noted.

[b] These figures are only approximate. The actual dose is at column "Total Mg. or C. c. injected."

F. Found dead at time noted.

Table XIII.—*Toxicity of arterenol hydrochloride*—Continued.

Number of mouse.	Mg base per gm.	Body weight.	Injected.			Died.
			Total.		Time.	
		Grams.	*Mg*	*C. c.*		
206	0.024	15.01	0.360	0.36	Jan. 9, 1.50 p. m.	
209	0.024	16.48	0.400	0.40	Jan. 9, 1.56 p. m.	
212	0.024	17.37	0.420	0.42	Jan. 9, 2.4 p. m.	
314	0.024	14.06	0.330	0.16	Jan. 20, 6.56 p. m.	
317	0.024	15.17	0.360	0.18	Jan. 20, 7.2 p. m.	
320	0.024	18.21	0.430	0.21	Jan. 20, 7.11 p. m.	
179a	0.024	13.51	0.320	0.32	Jan. 8, 12.37 p. m.	
121	0.028	14.43	0.404	0.40	Dec. 9, 5.45 p. m.	
185a	0.024	15.72	0.380	0.38	Jan. 8, 12.54 p. m.	
182a	0.024	18.78	0.450	0.45	Jan. 8, 12.44 p. m.	Jan. 11, 1.45 a. m. (W.).
183a	0.032	17.60	0.590	0.59	Jan. 8, 12.47 p. m.	
186a	0.032	12.88	0.410	0.41	Jan. 8, 12.56 p. m.	
198	0.032	20.23	0.650	0.65	Jan. 8, 1.27 p. m.	Jan. 9, 9.40 a. m. (F.).
201	0.032	15.25	0.490	0.49	Jan. 8, 1.33 p. m.	
204	0.032	13.75	0.440	0.44	Jan. 8, 1.58 p. m.	Jan. 9, 12.50 a. m. (W.).
207	0.032	16.60	0.530	0.53	Jan. 9, 1.53 p. m.	Jan. 10, 12.15 a. m. (W.).
210	0.032	13.40	0.430	0.43	Jan. 9, 1. 58 p. m.	
180a	0.032	17.59	0.590	0.59	Jan. 8, 12.39 p. m.	
213	0.032	14.14	0.45	0.45	Jan. 9, 2.6 p. m.	
315	0.030	21.06	0.63	0.31	Jan. 20, 6.58 p. m.	Jan. 20, 7.8 p. m.
318	0.030	16.21	0.48	0.24	Jan. 20, 7.6 p. m.	Jan. 22, 4.30 p. m.
321	0.030	19.84	0.59	0.29	Jan. 20, 7.13 p. m.	Jan. 22, 9 a. m. (W.).
199	0.040	18.12	0.72	0.72	Jan. 8, 1.28 p. m.	Jan. 9 (?), 10.45 a. m.
202	0.040	19.20	0.77	0.77	Jan. 8, 1.54 p. m.	Jan. 11, 10.30 a. m. (W.).
205	0.040	13.04	0.52	0.52	Jan. 8, 1.59 p. m.	
203a	0.040	13.67	0.54	0.54	Jan. 12, 4.4 p. m.	Jan. 12, 4.15 p. m.
208	0.040	15.58	0.62	0.62	Jan. 9, 1.55 p. m.	Jan. 11, 12.10 a. m. (W.).
211	0.040	17.44	0.70	0.70	Jan. 9, 2 p. m.	Jan. 11, 3 p. m. (W.).
214	0.040	17.87	0.72	0.72	Jan. 9, 2.8 p. m.	Jan. 10, 10.30 a. m. (W.).
305	0.040	20.12	0.80	0.40	Jan. 20, 6.7 p. m.	Jan. 20, 6.12 p. m.
308	0.040	19.52	0.78	0.36	Jan. 20, 6.15 p. m.	Jan. 20, 6.31 p. m.
311	0.040	15.02	0.60	0.30	Jan. 20, 6.24 p. m.	
316	0.040	14.48	0.58	0.29	Jan. 20, 7 p. m.	Jan. 20, 7.7 p. m.
319	0.040	15.39	0.61	0.30	Jan. 20, 7.9 p. m.	
205a	0.040	12.45	0.49	0.49	Jan. 12	
197a	0.040	14.07	0.56	0.56	Jan. 12	
200a	0.040	13.60	0.54	0.54	Jan. 12	
201a	0.040	14.21	0.57	0.57	Jan. 12	
322	0.040	17.90	0.71	0.35	Jan. 20, 7.15 p. m.	Jan. 21, 7.55 p. m. (F.).
181a	0.040	16.45	0.66	0.66	Jan. 8, 12.42 p. m.	
184a	0.040	14.17	0.56	0.56	Jan. 8, 12.52 p. m.	
187a	0.040	14.78	0.59	0.59	Jan. 8, 12.58 p. m.	
206a	0.040	12.87	0.51	0.51	Jan. 12	
209a	0.040	15.38	0.61	0.61	Jan. 12	
210a	0.040	13.45	0.53	0.53	Jan. 12	
212a	0.040	14.94	0.60	0.60	Jan. 12	
213a	0.040	13.54	0.55	0.55	Jan. 12	
217a	0.040	11.48	0.46	0.46	Jan. 12	
219a	0.040	12.63	0.50	0.50	Jan. 12	
222a	0.040	12.26	0.49	0.49	Jan. 12	

F. Found dead at time noted.

W. Time noted by watchmen.

TABLE XIII.—*Toxicity of arterenol hydrochloride*—Continued.

Number of mouse.	Mg. base per gm.	Body weight.	Injected.			Died.
			Total.		Time.	
		Grams.	*Mg.*	*C. c.*		
233	0.040	14.35	0.56	0.56	Jan. 12	
234	0.040	18.16	0.72	0.72	Jan. 12	
235	0.040	16.90	0.67	0.67	Jan. 12	
237	0.040	18.39	0.73	0.73	Jan. 12	
238	0.040	15.06	0.60	0.60	Jan. 12	
239	0.040	16.35	0.65	0.65	Jan. 12, 4.50 p. m.	Jan. 16, 8 a. m.
240	0.040	13.57	0.57	0.57	Jan. 12	
241	0.040	15.21	0.61	0.61	Jan. 12	
323	0.040	15.85	0.63	0.31	Mar. 15, 12.18 p. m.	Mar. 17, 6.45 a. m. (W.).
324	0.040	22.22	0.88	0.44	Mar. 15, 12.20 p. m.	Mar. 17, 12.20 p. m. (F.).
325	0.040	19.89	0.80	0.40	Mar. 15, 12.23 p. m.	
326	0.040	15.39	0.61	0.30	Mar. 15, 12.25 p. m.	Mar. 16, 6 a. m. (W.).
327	0.040	13.92	0.55	0.27	Mar. 15, 12.27 p. m.	
197b	0.060	14.53	0.87	0.43	Jan. 16, 11.1 p. m.	
200b	0.060	13.60	0.84	0.42	Jan. 16, 11.3 p. m.	
201b	0.060	14.51	0.87	0.43	Jan. 16, 11.5 p. m.	
306	0.060	17.44	1.05	0.52	Jan. 20, 6.11 p. m.	Jan. 20, 6.21 p. m.
309	0.060	23.06	1.38	0.69	Jan. 20, 6.18 p. m.	Jan. 20, 7.14 p. m.
312	0.060	19.03	1.14	0.57	Jan. 20, 6.27 p. m.	Jan. 23, 8 p. m.
328	0.060	21.37	1.28	0.64	Mar. 15, 12.33 p. m.	Mar. 15, 3.30 p. m. (F.).
329	0.060	16.54	0.99	0.49	Mar. 15, 12.40 p. m.	
330	0.060	19.07	1.14	0.57	Mar. 15, 12.42 p. m.	Mar. 17, 1.15 a. m. (W.).
331	0.060	20.23	1.21	0.60	Mar. 15, 12.44 p. m.	Mar. 17, 1.15 a. m. (W.).
332	0.060	21.68	1.30	0.65	Mar. 15, 12.45 p. m.	Mar. 15, 1 p. m.
205b	0.080	12.85	1.02	0.54	Jan. 16, 11.7 p. m.	
206b	0.080	13.62	1.09	0.54	Jan. 16, 11.14 p. m.	Jan. 17, 11.30 a. m. (W.).
209b	0.080	15.62	1.25	0.62	Jan. 16, 11.11 p. m.	
217b	0.080	13.09	1.05	0.52	Jan. 16, 11.24 p. m.	
219b	0.080	12.09	0.96	0.48	Jan. 16, 11.27 p. m.	
222b	0.080	12.89	1.03	0.51	Jan. 16, 11.29 p. m.	
307	0.080	14.85	1.19	0.59	Jan. 20, 6.13 p. m.	Jan. 22, 4 a. m. (F.).
310	0.080	16.14	1.28	0.64	Jan. 20, 6.20 p. m.	
313	0.080	15.50	1.24	0.62	Jan. 20, 6.31 p. m.	
333	0.080	12.96	1.03	0.51	Mar. 15, 12.54 p. m.	Mar. 15, 1.12 p. m.
334	0.080	21.85	1.74	0.87	Mar. 15, 12.57 p. m.	Mar. 20, 8 a. m. (W.).
335	0.080	18.11	1.44	0.72	Mar. 15, 12.59 p. m.	Mar. 19, 9.30 a. m. (W.).
336	0.080	13.68	1.09	0.54	Mar. 15, 1.10 p. m.	Mar. 19, 9.30 a. m. (W.).
337	0.080	11.81	0.94	0.47	Mar. 15, 1.6 p. m.	Mar. 15, 1.1 p. m.
338	0.100	14.45	1.44	0.72	Mar. 15, 1.8 p. m.	Mar. 17, 9 a. m. (F.).
339	0.100	13.16	1.31	0.65	Mar. 15, 1.10 p. m.	
340	0.100	18.56	1.85	0.92	Mar. 15, 1.13 p. m.	Mar. 15, 3.30 p. m. (F.).
341	0.100	12.02	1.20	0.60	Mar. 15, 1.14 p. m.	Mar. 15, 1.30 p. m. (F.).
342	0.100	13.01	1.30	0.65	Mar. 15, 1.17 p. m.	Mar. 16, 6 a. m. (W.).

W. Time noted by watchman.
F. Found dead at time noted.

PROTOCOLS TO EXPERIMENTS OF TABLE XIII.

It is important in making comparative studies that the experimental animal be healthy and free from the effects of previous injections and that the drugs employed show no signs of deterioration. Since some of the animals in Table XIII were injected more than once and some of the solutions were not used immediately, the following data is inserted to show this:

Mice Nos. 67 to 70, inclusive, 5 mg. arterenol hydrochloride crystals dissolved in 5 c. c. Ringer solution between 9 and 11 a. m. December 4, 1908.

Nos. 67, 68, 69, and 70 were previously injected November 30 with 0.22, 0.28, 0.42, and 0.61 mg., respectively, of homorenon hydrochloride.

Mice Nos. 107 to 111, inclusive, 6 mg. arterenol hydrochloride dissolved in 5 c. c. Ringer solution 9 a. m. December 9.

Nos. 117 to 121, inclusive, 6 mg. arterenol hydrochloride dissolved in 5 c. c. Ringer solution 11.30 a. m. December 9.

Nos. 179, 180, 181, 182, 183, 184, 185, 186, and 187, 6 mg. arterenol hydrochloride dissolved in 5 c. c. Ringer January 5 just before injecting; 179a, 180a, 181a, 182a, and 183a injected with a solution made up 3 p. m. January 7, for blood pressure experiment on January 7, whereas 184a, 185a, and 186a were injected with a solution made up in the same way 12.49, January 8.

Nos. 197, 198, 199, 200, and 202, 6 mg. arterenol hydrochloride dissolved in 5 c. c. Ringer, 12.49 p. m. January 8, 1909; 203, 204, and 205 similar solution made up 1.50 p. m. January 8; 197b, 200b, 201b, and 205b injected with a solution containing 12 mg. arterenol crystals dissolved in 5 c. c. Ringer, 10.57 p. m. January 16.

Nos. 206, 207, 208, 209, 210, 211, 212, 213, and 214, 6 mg. arterenol hydrochloride crystals dissolved in 5 c. c. Ringer 1.30 p. m. January 9, 1909; 206a, 209a, 210a, 212a, and 213a, 6 mg. crystals per 5 c. c. dissolved 3.53 p. m. January 12.

Nos. 206b, 209b, 210b, 212b, and 213b, 12 mg. arterenol HCl dissolved in 5 c. c. Ringer 10.57 p. m. January 16.

Nos. 217b, 219b, and 222b, 12 mg. arterenol hydrochloride crystals dissolved in 5 c. c. Ringer 10.57 p. m. January 16.

Nos. 233, 234, 235, 236, 237, 238, 239, 240, and 241, respectively, injected January 5 with 0.17, 0.30, 0.33, 0.24, 0.30, 0.32, 0.20, 0.23, and 0.32 mg. of arterenol hydrochloride and with the same drug again on January 8 with 0.32, 0.59, 0.66, 0.45, 0.59, 0.56, 0.38, 0.41, and 0.59 mg., respectively.

TOXICITY OF HOMORENON HYDROCHLORIDE.

It has been shown that it requires eighty times as much homorenon to produce the same rise of blood pressure in the dog as is caused by the same amount of natural l-adrenalin. If the relative toxicity of this compound as compared with l-adrenalin were in direct proportion to the relative vaso-constrictor action, then the lethal dose of homorenon ought to be 0.640 mg. per gram mouse, and as a matter of fact the lethal dose is somewhere between 0.568 and 0.711 mg. per gram mouse. It is true that a smaller dose than this sometimes kills and very rarely a mouse may survive as large a dose as 0.994 mg. per gram. But anything above 0.568 mg. so often kills that one is safe in saying that the lethal dose is slightly more than 0.568 and a little less than 0.711 mg. per gram body weight. According to Table XIV, then, the natural l-adrenalin is from eighty to eighty-eight times as toxic as ethyl-amino-aceto-catechol, commercially known as homorenon.

TABLE XIV.—*Toxicity of homorenon hydrochloride.*

Number of mouse.[a]	Mg. base per gm.[b]	Body weight.	Injected.			Died.
			Total.		Time	
		Grams.	*Mg.*	*C. c.*		
59	0.237	13.71	3.25	0.32	Dec. 4, 9.7 a. m.	
78	0.237	17.92	4.24	0.21	Dec. 7, 11.24 a. m.	
38	0.255	21.42	5.46	0.25	Dec. 2, 8.49 a. m.	
57	0.255	18.83	8.40	0.48	Dec. 3, 9.18 a. m.	
89	0.260	12.50	3.25	0.16	Dec. 7, 2.20 p. m.	
93	0.284	20.34	5.77	0.28	Dec. 7, 3.44 p. m.	
296	0.284	13.31	3.78	0.07	Jan. 20, 4.39 p. m.	
299	0.284	21.15	6.00	0.12	Jan. 20, 4.46 p. m.	Jan. 23, 8 a. m. (W.).
302	0.284	15.32	4.30	0.08	Jan. 20, 4.53 p. m.	
60	0.296	20.89	6.18	0.62	Dec. 4, 9.10 a. m.	
90	0.347	18.28	6.35	0.31	Dec. 7, 2.24 p. m.	
64	0.356	14.70	5.23	0.52	Dec. 4, 9.14 a. m.	
79	0.356	22.32	7.94	0.39	Dec. 7, 11.26 a. m.	Dec. 10 (F.).
94	0.426	19.32	8.23	0.41	Dec. 7, 3.46 p. m.	
122	0.426	21.91	9.33	0.46	Dec. 10, 2.11 p. m.	
127	0.426	16.97	7.23	0.36	Dec. 10, 2.24 p. m.	
132	0.426	17.24	7.35	0.36	Dec. 10, 2.40 p. m.	
137	0.426	12.33	5.50	0.27	Dec. 10, 2.55 p. m.	
297	0.426	21.94	9.34	0.18	Jan. 20, 4.41 p. m.	Jan. 22, 4 p. m. (F.).
300	0.426	18.90	8.00	0.16	Jan. 20, 4.48 p. m.	
303	0.426	18.52	7.80	0.15	Jan. 20, 4.55 p. m.	
260	0.427	14.30	6.15	0.12	Jan. 16, 3.50 p. m.	
263	0.427	17.30	7.38	0.14	Jan. 16, 3.58 p. m.	
266	0.427	22.78	9.73	0.19	Jan. 16, 4.5 p. m.	
269	0.427	18.57	7.93	0.16	Jan. 16, 9.49 p. m.	Jan. 16, 10.14 p. m.
272	0.427	19.13	8.17	0.16	Jan. 16, 10 p. m.	Jan. 16, 11.30 p. m.
275	0.427	16.94	7.23	0.14	Jan. 16, 10.6 p. m.	
287	0.427	15.05	6.40	0.13	Jan. 20, 3.8 p. m.	Jan. 20, 3.39 p. m.
290	0.427	15.63	6.70	0.13	Jan. 20, 3.23 p. m.	Jan. 24, 3.36 p. m.
293	0.427	16.36	6.98	0.14	Jan. 20, 3.29 p. m.	
278	0.430	14.92	6.40	0.13	Jan. 16, 11.46 p. m.	
279	0.430	19.02	8.17	0.16	Jan. 16, 11.49 p. m.	Jan. 16, 11.58 p. m.
280	0.430	16.79	7.18	0.14	Jan. 16, 11.50 p. m.	
281	0.430	15.04	6.45	0.13	Jan. 16, 11.52 p. m.	
282	0.430	18.52	7.95	0.16	Jan. 16, 11.54 p. m.	Jan. 17, 11.30 a. m. (W.).
283	0.430	15.02	6.45	0.13	Jan. 16, 11.56 p. m.	
284	0.430	13.43	5.76	0.11	Jan. 16, 11.58 p. m.	
285	0.430	15.83	6.79	0.13	Jan. 16, 12.1 p. m.	Jan. 17, 4 p. m. (W.).
91	0.434	15.10	6.55	0.32	Dec. 7, 2.26 p. m.	
80	0.474	19.30	9.14	0.45	Dec. 7, 11.29 a. m.	
97	0.474	14.20	6.73	0.33	Dec. 8, 9.42 a. m.	
39	0.518	17.16	8.89	0.40	Dec. 2, 9.7 a. m.	
95	0.568	14.22	8.07	0.40	Dec. 7, 3.50 p. m.	
123	0.568	21.71	12.32	0.61	Dec. 10, 2.14 p. m.	
128	0.568	13.46	7.66	0.38	Dec. 10, 2.27 p. m.	
133	0.568	13.74	7.81	0.39	Dec. 10, 2.42 p. m.	
138	0.568	12.36	7.02	0.35	Dec. 10, 2.58 p. m.	
251	0.568	20.88	11.86	0.23	Jan. 16, 3.15 p. m.	

[a] Mice not marked "W" or "F" were observed by the author to die at the time noted.

[b] This column approximate dose only; actual dose in "total mg. and c. c. columns."

W. Time noted by watchman.

F. Found dead at time noted.

TABLE XIV.—*Toxicity of homorenon hydrochloride*—Continued.

Number of mouse.	Mg. base per gm.	Body weight.	Injected.			Died.
			Total.		Time.	
		Grams.	*Mg.*	*C. c.*		
254	0.568	18.20	10.37	0.30	Jan. 16, 3.36 p. m.	Jan. 16, 3.55 p. m.
257	0.568	20.66	11.74	0.23	Jan. 16, 3.40 p. m.	Jan. 16, 3.47 p. m.
261	0.568	20.06	11.40	0.22	Jan. 16, 3.51 p. m.	
264	0.568	21.64	12.27	0.24	Jan. 16, 3.59 p. m.	Jan. 16, 4.15 p. m.
267	0.568	19.76	11.22	0.22	Jan. 16, 4.8 p. m.	
270	0.568	19.60	11.13	0.22	Jan. 16, 9.56 p. m.	
273	0.568	17.84	10.47	0.21	Jan. 16, 10.12 p. m.	
276	0.568	19.22	10.91	0.22	Jan. 16, 10.8 p. m.	Jan. 16, 10.21 p. m.
291	0.568	17.83	10.1	0.20	Jan. 20, 3.25 p. m.	Jan. 20, 3.49 p. m.
294	0.568	18.35	10.4	0.21	Jan. 20, 3.31 p. m.	Jan. 20, 3.57 p. m.
298	0.568	19.08	10.78	0.21	Jan. 20, 4.44 p. m.	Jan. 20, 5 p. m.
301	0.568	14.20	8.00	0.16	Jan. 20, 4.49 p. m.	Jan. 20, 5.20 p. m.
304	0.568	17.13	9.7	0.19	Jan. 20, 4.57 p. m.	
288	0.583	14.73	8.6	0.17	Jan. 20, 3.12 p. m.	Jan. 20, 3.57 p. m.
81	0.593	17.62	10.44	0.52	Dec. 7, 11.32 a. m.	Dec. 7, 12.5 p. m.
98	0.593	14.12	8.37	0.41	Dec. 8, 9.45 a. m.	
99	0.711	17.05	12.12	0.60	Dec. 8, 9.47 a. m.	
124	0.710	17.07	12.11	0.60	Dec. 10, 2.17 p. m.	
129	0.710	12.97	9.21	0.46	Dec. 10, 2.31 p. m.	
134	0.710	20.10	14.27	0.70	Dec. 10, 2.45 p. m.	
139	0.710	18.11	12.86	0.64	Dec. 10, 3 p. m.	
215	0.711	19.05	13.53	0.27	Jan. 16, 3.17 p. m.	Jan. 16, 3.52 p. m. (F.).
255	0.711	23.65	16.79	0.33	Jan. 16, 3.38 p. m.	Jan. 16, 4.10 p. m. (F.).
258	0.711	19.09	13.56	0.27	Jan. 16, 3.44 p. m.	
262	0.711	19.80	14.05	0.28	Jan. 16, 3.54 p. m.	
265	0.711	25.00	17.75	0.35	Jan. 16, 4.3 p. m.	Jan. 16, 4.7 p. m.
268	0.711	22.58	16.03	0.32	Jan. 16, 4.10 p. m.	Jan. 16, 4.20 p. m.
271	0.711	19.47	13.84	0.27	Jan. 16, 9.58 p. m.	Jan. 16, 11.30 p. m.
274	0.711	15.59	11.08	0.22	Jan. 16, 10.4 p. m.	Jan. 16, 10.16 p. m.
277	0.711	19.80	14.07	0.28	Jan. 16, 10.10 p. m.	Jan. 16, 10.21 p. m.
289	0.711	15.13	10.7	0.21	Jan. 20, 3.21 p. m.	
292	0.711	18.56	13.20	0.26	Jan. 20, 3.26 p. m.	Jan. 20, 3.47 p. m.
295	0.711	16.86	12.00	0.24	Jan. 20, 3.35 p. m.	Jan. 20, 3.51 p. m.
40	0.777	19.47	15.13	0.69	Dec. 2, 9.5 a. m.	Dec. 2, 10.25 a. m.
100	0.830	13.26	11.01	0.55	Dec. 8, 9.50 a. m.	
125	0.852	16.37	13.95	0.69	Dec. 10, 2.19 p. m.	
130	0.852	18.28	15.57	0.78	Dec. 10, 2.33 p. m.	
135	0.852	19.37	16.50	0.81	Dec. 10, 2.48 p. m.	
140	0.852	17.55	14.95	0.74	Dec. 10, 3.3 p. m.	Dec. 10, 3.35 p. m.
170	0.853	12.89	10.99	0.20	Jan. 5, 3 p. m.	
173	0.853	12.57	12.38	0.24	Jan. 5, 3.5 p. m.	Jan. 5, 3.29 p. m.
176	0.853	14.37	12.26	0.24	Jan. 5, 3.14 p. m.	Jan. 5, 3.31 p. m.
245	0.853	20.05	17.04	0.34	Jan. 16, 1.25 p. m.	Jan. 16, 1.45 p. m.
248	0.853	22.60	19.21	0.38	Jan. 16, 1.34 p. m.	Jan. 16, 1.57 p. m.
253	0.853	18.05	15.34	0.30	Jan. 16, 3.19 p. m.	Jan. 16, 4 p. m.
256	0.853	19.17	16.29	0.32	Jan. 16, 3.39 p. m.	Jan. 16, 4.12 p. m.
259	0.853	14.71	12.50	0.25	Jan. 16, 3.46 p. m.	Jan. 16, 4 p. m.
101	0.949	14.14	13.42	0.53	Dec. 8, 9.53 a. m.	Dec. 8, 11 a. m.
126	0.994	12.15	12.08	0.60	Dec. 10, 2.22 p. m.	
131	0.994	15.73	15.64	0.78	Dec. 10, 2.36 p. m.	Dec. 10, 3.35 p. m.
136	0.994	14.11	14.02	0.70	Dec. 10, 2.50 p. m.	Dec. 10, 3.15 p. m.

F. Found dead at time noted.

TABLE XIV.—*Toxicity of homorenon hydrochloride*—Continued.

Number of mouse.	Mg. base per gm.	Body weight.	Injected.			Died.
			Total.		Time.	
		Grams.	*Mg.*	*C. c.*		
141	0.994	19.43	19.31	0.96	Dec. 10, 3.6 p. m.	Dec. 10, 3.30 p. m.
171	0.994	14.50	14.41	0.28	Jan. 5, 3.2 p. m.	Jan. 5, 3.20 p. m.
174	0.994	13.20	13.12	0.26	Jan. 5, 3.9 p. m.	Jan. 5, 3.31 p. m.
177	0.944	13.76	13.68	0.28	Jan. 5, 3.15 p. m.	Jan. 5, 3.38 p. m.
243	0.994	20.64	20.52	0.41	Jan. 16, 1.21 p. m.	Jan. 16, 1.28 p. m.
246	0.994	22.59	22.33	0.44	Jan. 16, 1.27 p. m.	Jan. 16, 1.39 p. m.
249	0.994	18.31	18.12	0.36	Jan. 16, 1.36 p. m.	Jan. 16, 1.55 p. m.
41	1.038	19.47	20.21	0.92	Dec. 2, 9 a. m.	Dec. 2, 9.55 a. m.
172	1.137	20.93	23.80	0.48	Jan. 5, 3.4 p. m.	Jan. 5, 3.15 p. m.
175	1.137	17.00	19.53	0.38	Jan. 5, 3.10 p. m.	Jan. 5, 3.29 p. m.
178	1.137	13.42	14.78	0.30	Jan. 5, 3.20 p. m.	Jan. 5, 3.47 p. m.
244	1.137	15.22	17.20	0.34	Jan. 16, 1.22 p. m.	Jan. 16, 2 p. m.
247	1.137	17.78	20.11	0.40	Jan. 16, 1.29 p. m.	Jan. 16, 1.39 p. m.
250	1.137	22.37	25.31	0.50	Jan. 16, 1.37 p. m.	Jan. 16, 1.50 p. m.

PROTOCOLS TO EXPERIMENTS OF TABLE XIV.

The solutions of homorenon hydrochloride used in the different toxicity experiments were made up as follows:

Nos. 37 to 41, inclusive, 66 mg. homorenon hydrochloride crystals dissolved in 3 c. c. of Ringer solution about 8.30 a. m. December 2.

Nos. 47 to 51, inclusive, 20 mg. of crystals dissolved in 2 c. c. of Ringer solution about 9 a. m. December 3.

Nos. 57 to 61, inclusive, 25 mg. crystals dissolved in 2.5 c. c. of Ringer solution 11 a. m. December 7.

Nos. 87, 88, 89, 90, and 91 injected December 4 with 0.89, 1.90, 3.25, 6.18, and 5.23 mg., respectively, of homorenon, and 92, 93, 94, 95, and 96 were each, respectively, injected on December 2 with 0.038 mg. of adrenalin, 0.125, 0.225, 0.518, and 1.45 mg. homorenon.

Nos. 97 to 101, inclusive, 20 mg. crystals dissolved in 1 c. c. of Ringer solution.

Nos. 122 to 141, inclusive, 20 mg. per 1 c. c. of Ringer solution made up 2 p. m. December 10.

Nos. 122, 123, 124, 125, and 126 had been injected with 0.021, 0.042, 0.069, 0.081, and 0.082 mg., respectively, of adrenalin base December 4, and again on December 7 with 0.040, 0.080, 0.103, 0.128, and 0.130 mg., respectively, of the same drug.

Nos. 127, 128, 129, 130, and 131 were injected December 4 with 0.890, 1.90, 3.25, 6.18, and 5.23 mg., respectively, of homorenon, and again on December 7 with 1.33, 2.25, 3.25, 6.35, and 6.55 mg., respectively, of the same drug.

No. 132 was injected December 2 with 0.038 mg. of adrenalin base and on December 7 with 2.48 mg. homorenon, No. 133 on December 4 and 7, respectively, with 0.890 and 2.48 mg. of homorenon, while Nos. 134, 135, and 136 were each injected on December 2 with 0.125, 0.225, and 0.518 mg., respectively, of homorenon, and again on December 7 with 5.77, 8.23, and 8.07 mg., respectively, of the same drug.

Finally mice Nos. 139, 140, and 141, respectively, had been injected on December 3 with 0.554, 0.876, and 2.688 mg., and again on the 7th of December with 2.08, 1.21, and 9.14 mg. of homorenon.

Nos. 170 to 178, inclusive, 250 mg. of homorenon hydrochloride crystals dissolved in 5 c. c. of Ringer, about 2.45 p. m. January 5, 1909.

Nos. 242 to 250, inclusive, and 267 and 268, 250 mg. of crystals dissolved in 5 c. c. Ringer solution about 1 p. m. January 16.

Nos. 251 to 266 and 269 to 286, inclusive, 250 mg. of crystals dissolved in 5 c. c. Ringer solution 3 p. m. January 16 for each set of nine.

Nos. 287 to 304, inclusive, 250 mg. of crystals dissolved in 5 c. c. Ringer solution 2.30 p. m. January 20, 1909.

MEASUREMENT OF MYDRIASIS IN THE FROG'S EXCISED BULBUS.[a]

As is well known the dilator mechanism of the frog's eye is quite sensitive to adrenalin and not a little emphasis has been laid upon the delicacy with which it reacts to minute traces of this and certain other derivatives of the catechol group. Because of this mydriatic action, the apparent simplicity of the technique involved, and the availability of experimental animals, the enucleated eye of the frog seems to furnish a suitable method for standardizing adrenalin solutions.

Meltzer was the first to call attention to this method and nearly a year later Ehrmann, acting upon Meltzer's suggestion, published some very interesting experiments with the excised frog's eye. In Ehrmann's suggestive paper it is not only shown that adrenalin solutions of different concentration call forth varying degrees of mydriasis, but it leads one to suppose that the enucleated frog's eye is sensitive to such minute quantities of adrenalin as may occur in the blood drawn from the vena cava. Taking advantage of the sensitiveness of the frog's eye he also uses this method as a means for demonstrating the activity of adrenalin still in the blood of injected animals bled immediately after the fall of blood pressure that invariably follows the initial effect of this drug.[b] Just how reliable these conclusions are remains to be decided by future experiments. Cameron, in his paper on methods of standardizing suprarenal preparations, states that the method is not only tedious but very unreliable, since frogs vary in their response to adrenalin.

Various factors do influence the reaction of the iris to adrenalin. Perhaps the most important ones are: (1) Conditions that interfere with the frog's normal metabolism and nutrition, (2) the varying intensity of light to which the eye is exposed, (3) the temperature of the medium surrounding the enucleated bulbus, (4) injuries to the coats of the bulbus that may result in lowering the intra-ocular tension and alter the rate of diffusion, and, finally, (5) mechanical stimuli. One of the main problems of this method is to devise a technique that minimizes the chief sources of error.

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[a] The section on the pupil was completed November 15, 1908, but publication was delayed so that the comparative study herein contained might be made.

[b] Dryer as early as 1897-1899 showed that adrenalin is secreted into the circulation after stimulation of the splanchnic nerve. This excellent piece of work has not received the attention of German writers that it should, especially since Dryer is one of the first to prove conclusively that stimulation of the splanchnic causes an increased secretion by the adrenal glands.

As is well known, the frog's pupil is an elliptical-like opening the long axis of which is almost parallel to the long axis of the body. On the lower margin of the pupil, almost in line with the short axis of the ellipse, there is a small notch, whereas the upper margin, though usually continuous, is sometimes indented by a less conspicuous notch. In measuring the eye, especially when widely dilated, these notches furnish convenient points of reference in locating the short axis. The long axis in the undilated eye is of course easily located by reason of the shape of the anterior and posterior margins of the pupil.

In orienting the bulbus previous to measuring the size of the pupil it is necessary to have a suitable container. Glass vessels of about 3 c. c. capacity were used, made from hard bacteriological test tubes 17 mm. in diameter, the edges being ground smooth so that it might be sealed with a cover slip to avoid evaporation.

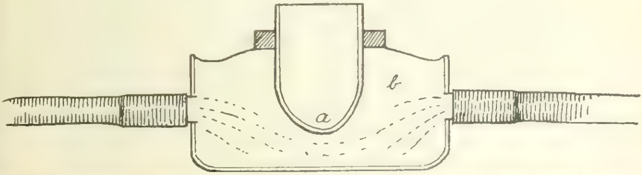


FIG. 1. Water bath. *a*, Glass container, described in text. *b*, water bath through which a steady stream of water from the temperature regulator is kept flowing.

Except in a few experiments described later, special care was taken to avoid the changes in temperature and light values that occur in a laboratory lighted by direct or diffuse sunlight. To this end a dark room was fitted up with a 16-candlepower incandescent light so arranged that the excised bulbi could be lighted from a given distance, thus making it possible to maintain a light of constant intensity. Of course at a distance of 10 inches such a light generates enough heat to warm 2 c. c. of solution to 30° or more, depending upon the room temperature. In order to avoid not only such high temperatures, but more especially fluctuations in the same, the water bath, indicated in figure 1, was devised. It is constructed in such a way that the glass container rests in a current of water, the temperature of which is so regulated that the temperature of the solution in the container can also be kept constant.

Preliminary experiments demonstrated that a mere naked eye observation of the changes in size of the pupil is of little value, and so an instrument of precision was sought that would enable the observer to express in millimeters the exact length of the long and short axes of the pupil. The device which proved to be most satisfactory was made for me by Messrs. Gaertner & Co., of Chicago. Their "simple comparator" was modified by the author to meet the needs

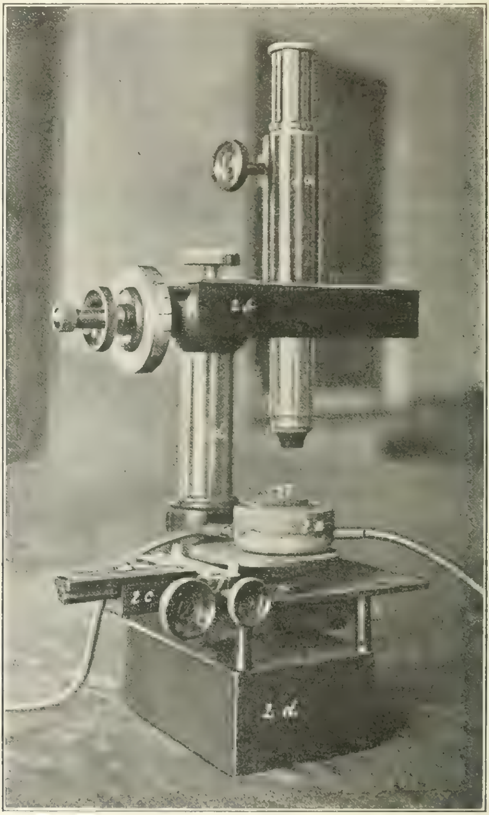
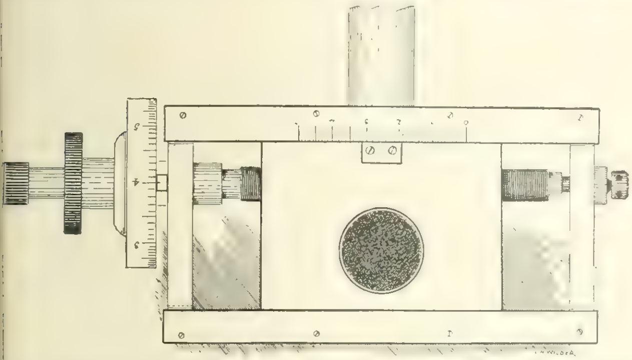


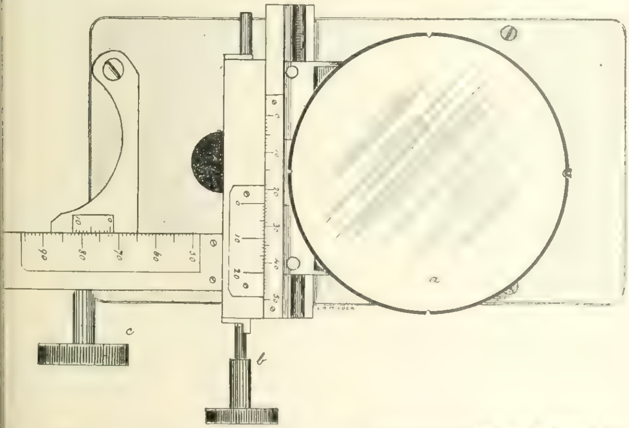
FIG. 2.—Pupilometer. *2a*, Reading microscope with cross wires; *2b*, micrometer slide (see fig. 3); *2c*, adjustable substage (see fig. 4); *2d*, support; *2e*, water bath (see fig. 1). Temperature regulators and light omitted for sake of clearness.

of the present series of experiments. (See fig. 2.) The essential parts of the apparatus are, (1) a reading microscope with cross wires in the eyepiece (fig. 2a), (2) a micrometer slide (fig. 2b and fig. 3), (3) an djustable stage (fig. 2e and fig. 4), and, finally, (4) a support (fig. 2d). he cross wire in the eyepiece furnishes a point of reference, and in aking a reading the eye is first oriented so as to bring its optical



IG. 3.—Micrometer slide. Micrometer screw has a diameter of 10 mm., pitch of 0.5 mm. The micrometer head has a diameter of 50 mm. and is divided into 100 parts, each division reading to 0.005 mm.

xis parallel with the axis of the microscope. Then by means of a oarse adjustment the desired edge of the pupil is brought into line



G. 4. Adjustable substage. a, Revolving support, upon which the [illegible] rack and pinion providing quick adjustment for centering [illegible]

ith the cross wire and the long axis of the pupil measured, the bject stage is rotated to 90° and the short axis measured. In this ay a given eye can be measured many times without disturbing it by jars or other forms of mechanical stimulation except when it is necessary to change the solution in the container.

Since the unit that most concerns us in this paper is the time required by the pupil to dilate to a maximum it becomes necessary to explain a few points in this connection. (1) In some eyes the long axis is the first to reach a maximum length; at other times it is the short axis. (2) The axis first to reach a maximum length usually shortens while the other axis is in the process of elongating. It is only occasionally that both axes reach their maximum length simultaneously, or that the one waits upon the other. Hence it often becomes difficult to determine the exact moment at which the eye is most widely dilated unless the absolute area of the pupil be calculated. This is too tedious a process, so as a rule one of the following methods is used: (1) The mean of the time required for the two axes to reach their greatest length; (2) the moment of maximum dilation determined by the first axis reaching its greatest length or turning point. Unless otherwise stated, the last method, though less accurate, is the one used throughout the paper.

I always endeavored to choose frogs of the same variety, that weighed not less than 17 grams nor more than 30. The head was cut off just back of the eyes and the bulbi removed from their sockets at once. In removing the eyeball undue pressure was avoided and care taken not to cut the sclerotic coat. The bulbi were immediately dropped into a container of Ringer solution, observed and measured. When necessary to change the solution, it was carefully pipetted off and a fresh solution of nearly the same temperature substituted.

In spite of all precautions certain eyes will dilate and remain dilated in the Ringer solution for an unusual length of time. When this occurs they should be rejected. Furthermore, eyes from frogs of low vitality seem to be more susceptible to adrenalin than those of healthy, vigorous animals. This source of variation is the most difficult to overcome, and perhaps accounts for many of the variations that occur in experiments of this nature.

ABLE XV.—*Mydriatic action of adrenalin upon the frog's eye when exposed to bright sunlight.*

Experiment 17, July 22, 1908. Rana pipiens. Weight about 17 grams. Killed [illegible] .18. The solutions made from P. D. and Co.'s 1:1000 [illegible]. [illegible] 18° to 32.5° C., and the brightness of the light varied from the bright [illegible] 4.30 p. m. of a clear July day. (−) always indicates decrease in size, i. e., constriction of the pupil.

Ringer. A.			Adrenalin solution + Ringer 1:5,000,000 B.			Adrenalin solution + Ringer 1:2,500,000 C.			Adrenalin solution + Ringer 1:625,000 D.			Ringer. E.			
ime.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	C.
. m.	M. m.	M. m.	A. m.	M. m.	M. m.	A. m.	M. m.	M. m.	A. m.	M. m.	M. m.	A. m.	M. m.	M. m.	
0.20	1.4	2.6	10.22	1.5	2.5	10.24	1.5	2.5	10.02	1.5	2.8	10.30	1.5	2.6	29.8
	.....	.....	10.33	In adr.		10.33	In adr.		10.33	In adr.					
0.33	[a] 1.1	2.4	10.30	1.1	2.4	10.40	1.3	2.4	10.45	1.4	2.6	10.47	1.5	2.8	30.0
	[b] −.3	− .2		− .4	− .1		− .2	− .1		− .1	− .2		.0	+ .2	
0.50	1.1	2.4	10.52	1.4	2.4	10.54	1.3	2.0	10.50	1.1	2.5	11	1.1	2.5	30.5
	− .3	− .2		− .1	− .1		− .2	− .5		− .4	− .3		− .4	− .1	
1.00	1.2	2.3	11.02	1.2	2.2	11.04	1.2	2.0	11	1.0	2.4	11.20	1.0	2.1	31.3
	− .2	− .3		− .3	− .3		− .3	−.5		− .5	− .4		− .5	− .5	
1.40	1.1	2.1	11.42	1.1	2.1	11.44	1.5	2.1	11.04	1.1	2.4	11.50	1.1	2.4	32.5
	− .3	− .5		− .4	− .4		.0	− .4		− .4	− .4		[illegible]	− .2	
P. m.			P. m.			P. m.			P. m.			P. m.			
2.20	1.2	2.2	12.32	1.2	2.3	12.24	1.5	2.3	12.28	1.2	2.4	12.30	1.2	2.4	31.8
	− .2	− .4		− .3	− .2		.0	− .2		− .3	− .4		− .3	− .4	
2.53	1.3	2.3	12.55	1.4	2.4	12.56	2.0	2.5	12.59	1.1	2.4	1.1	1.1	2.4	31.0
	— .1	− .3		− .1	− .1		+ .5	.0		− .4	− .4		− .4	− .2	
2.04	1.4	2.7	2.06	1.5	2.3	2.08	2.4	2.6	2.12	1.4	2.5	2.14	1.5	2.6	31.0
	.0	+ .1		.0	− .2		+ .9	+ .1		− .1	− .3		.0	.0	
3.14	1.6	2.6	3.16	1.5	2.8	3.18	2.6	2.8	3.22	1.6	2.5	3.24	1.7	2.5	31.5
	+ .2	.0		.0	+ .3		+1.1	+ .3		+ .1	− .3		+ .2	− .1	
4.14	1.9	2.6	4.16	1.6	2.5	4.18	2.7	2.9	4.22	1.6	2.6	4.24	1.9	2.6	30.6
	+ .5	.0		+ .1	.0		+1.2	+ .4		+ .1	− .2		+ .4	.0	

[a] First line in each group indicates actual length of axes in millimeters.

[b] Second line in each group shows change in length of axes.

Other investigators in their experiments with adrenalin and the excised bulbus used sunlight, which as is well known varies greatly in intensity. Since the stimulating action of adrenalin is supposed to counteract the stimulating action of light it would seem of the greatest importance to use light stimuli of a constant value and of optimum intensity. This, I believe, I have accomplished by the use of electric light which, so far as I know, has never been used before in experiments of this nature.

In the first place the constricting effect of very bright sunlight is usually greater than can be overcome by the dilating effects of the adrenalin present in a 1:5,000,000 or even in a 1:625,000 solution. Therefore, the readings from the start have a negative sign, that is, the pupil constricts. It is only after remaining in the solution two or three hours that signs of dilation appear, and even then there is no positive evidence in favor of the adrenalin causing this dilation.

The readings in columns A and E of Table XV are from eyes in Ringer solution. The maximum constriction is −.3 for the long axis and −.5 mm. for the short axis of A; −.5 by −.5 mm. for E. Later, as the light grows dimmer and the temperature lower, the same eyes dilate making the difference in their sizes a little after 4 p. m. from that when first measured for A +.5 by .0 mm.; and for E +.4 by .0 mm., the pupils at the end being slightly larger than when first measured. It is true that C shows greater dilation than this, even as much as +1.2 by +.4, but D in a much stronger solution of adrenalin than C dilates less. In the light of simliar sets of experiments, I am inclined not to stress the importance of adrenalin in bringing about the dilation in B, C, or D, for if they had been in Ringer solution alone they would without doubt have reacted in much the same way. On the whole I found sunlight unsatisfactory. With few exceptions, eyes under its influence in weak solution constricted, and if there were dilation later, it was only after exposure to Ringer solution and at a temperature which my previous experience with salt solutions has shown to be positively injurious to all muscle tissue.

Mechanical stimuli are likewise an important factor in bringing about dilation of the pupil. Due care must be taken in changing the solution and in orienting the pupil, especially at such temperatures as render smooth muscle unusually irritable.

TABLE XVI. *The effect of mechanical stimuli upon the pupil of the excised bulbus, kept at a constant temperature and lighted by artificial light.*

Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.
A. m.	*M. m.*	*M. m.*	*A. m.*	*M. m.*	*M. m.*	*A. m.*	*M. m.*	*M. m.*
10.12	2.235	2.055	11.51	1.975	0.945	1.12	1.670	1.170
10.25	2.220	1.860	11.58	1.670	1.165	1.15	1.710	1.200
10.48	1.835	1.440	*P. m.*			1.20	1.825	1.400
11.18	1.535	0.970	12.2	1.735	1.290	1.26	1.950	1.645
11.29	1.515	0.960	12.11	1.695	1.240	1.36	1.935	1.610
11.46	1.490	0.900	12.37	1.635	1.045	1.48	1.850	1.405
11.48	By means of a capillary pipette the eye is gently pipetted about in its bath and then oriented as usual.		12.48	1.650	1.150	2.8	1.815	1.310
			1.2	1.635	1.080	2.27	1.800	1.370
			1.9	Ringer pipetted off and 2 c. c. of a 1:3,125,000 solution of adrenalin added.				

Table XVI illustrates in a general way the reaction of the pupil to mechanical stimuli. Soon after being placed in the container the eye dilates to a maximum (10.48), then constricts to a minimum, in which condition it remains for a long time if all influences be kept constant. But by gently pipetting the eye about as is done (11.48 a. m.) it dilates, sometimes slowly, but in this case rapidly, after which it again constricts. The experiment was continued (1.9) by replacing the Ringer solution with a very dilute solution of adrenalin of the same temperature, disturbing the eye to about the same extent as before. This again caused dilation similar to that following the mechanical stimuli just described. At first sight this last dilation might easily be attributed to the adrenalin since the eye not only dilates more slowly but the dilation persists. Other observations, however, lead me to conclude that the adrenalin has but little action and that which it has is exerted in supplementing the mechanical stimuli and perhaps in retarding the after constriction.

Since ordinary sunlight is unsatisfactory for quantitative work of this nature, artificial light was substituted. If the proper intensity be used and the eyes observed at 22 to 23° C. then, instead of constriction, dilation may occur with very dilute solutions. The dilation sometimes present with a concentration of 1 of adrenalin to 625,000 of Ringer or with weaker solutions is, however, not at all suited for quantitative determinations.

TABLE XVII.—*Mydriatic action of natural l-adrenalin.*

EXCISED FROG'S EYE.

Experiment 39a. 1: 2,000.			Experiment 41a. 1: 5,000.			Experiment 55b. 1: 25,000.		
Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.
A. m.	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*
10. 12	[a] 2. 135	[a] 1. 700	3. 56	[a] 2. 010	[a] 1. 430	2. 10	[a] 1. 760	[a] 1. 660
10. 15	[a] 2. 115	[a] 1. 690	4. 5	[a] 2. 055	[a] 1. 650	2. 32	[a] 2. 030	[a] 1. 840
10. 20	[a] 2. 070	[a] 1. 720	....	....	...	3. 25	[a] 1. 765	[a] 1. 435
10. 21	[a] 2. 125	[a] 1. 730	....	....	...	3. 30	[a] 1. 730	[a] 1. 510
10. 26	[a] 2. 135	[a] 1. 740	4. 8	[a] 2. 100	[a] 1. 650	3. 42	[a] 1. 790	[a] 1. 595
10. 27	In adrenalin.		4. 9	In adrenalin.		3. 44	In adrenalin.	
10. 29	[b] 2. 185	[b] 1. 750	4. 11	[b] 2. 105	[b] 1. 805	3. 46	[b] 1. 900	[b] 1. 795
2	[c] 0. 050	[c] 0. 010	2	[c] 0. 005	[c] 0. 155	2	[c] 0. 110	[c] 0. 200
10. 31	[b] 2. 250	[b] 1. 935	4. 13	[b] 2. 510	[b] 2. 305	3. 48	[b] 1. 990	[b] 1. 940
4	[c] 0. 115	[c] 0. 195	4	[c] 0. 410	[c] 0. 655	1	[c] 0. 200	[c] 0. 345

[a] Actual length of axis measured in 2 c. c. of Ringer solution at 23° C.

[b] Actual length of axis at time measured.

[c] Change in length of axis after the eye is exposed to 2 c. c. of adrenalin solution for the number of minutes indicated to left in time column.

TABLE XVII. *Mydriatic action of natural l-adrenalin*—Continued.

EXCISED FROG'S EYE—Continued.

Experiment 39a. 1:2,000.				Experiment 41a. 1:5,000.				Experiment 55b. 1:25,000.		
Time.	Long axis.		Short axis.	Time.	Long axis.		Short axis.	Time.	Long axis.	Short axis.
10.27	In adrenalin.			4.9	In adrenalin.			3.44	In adrenalin.	
A. m.	*M. m.*		*M. m.*	*P. m.*	*M. m.*		*M. m.*	*P. m.*	*M. m.*	*M. m.*
10.34	[a] 2.515		[a] 2.440	4.15	[a] 2.700		[a] 2.510	3.54	[a] 2.100	[a] 2.065
7	[b] 0.380	23.0°	[b] 0.700	6	[b] 0.600	23.8°	[b] 0.860	6	[b] 0.310	[b] 0.470
10.36	[a] 2.830	C.	[a] 2.685	4.18	[a] 2.750	C.	[a] 2.640	3.52	[a] 2.235	[a] 2.150
9	[b] 0.695		[b] 0.945	9	[b] 0.650		[b] 0.990	8	[b] 0.445	[b] 0.555
10.38	[a] 2.950		[a] 2.785	4.21	[a] 2.850		[a] 2.760	3.54	[a] 2.360	[a] 2.245
11	[b] 0.815		[b] 1.045	12	[b] 0.750		[b] 1.110	10	[b] 0.570	[b] 0.650
10.40	[a] 2.950		[a] 2.830	4.23	[a] 2.860		[a] 2.800	3.58	[a] 2.520	[a] 2.350
13	[b] 0.815		[b] 1.090	14	[b] 0.760		[b] 1.150	14	[b] 0.730	[b] 0.755
10.42	[a] 2.950		[a] 2.830	4.25	[a] 2.840		[a] 2.780	4.00	[a] 2.555	[a] 2.400
15	[b] 0.815		[b] 1.090	16	[b] 0.740		[b] 1.130	16	[b] 0.765	[b] 0.805
10.44	[a] 2.950	23.1°	[a] 2.830	4.27	[a] 2.840		[a] 2.800	4.3	[a] 2.610	[a] 2.430
17	[b] 0.815	C.	[b] 1.090	18	[b] 0.710		[b] 1.110	19	[b] 0.820	[b] 0.835
10.47	[a] 2.970		[a] 2.830	4.35	[a] 2.775		[a] 2.820	4.6	[a] 2.665	[a] 2.410
20	[b] 0.835		[b] 1.090	26	[b] 0.675		[b] 1.170	22	[b] 0.875	[b] 0.815
10.50	[a] 2.935		[a] 2.830	4.40	[a] 2.640		[a] 2.840	4.8	[a] 2.695	[a] 2.425
23	[b] 0.800		[b] 1.090	31	[b] 0.540		[b] 1.190	24	[b] 0.905	[b] 0.830
10.52	[a] 2.900		[a] 2.815	4.45	[a] 2.600		[a] 2.800	4.11	[a] 2.705	[a] 2.425
25	[b] 0.765		[b] 1.075	36	[b] 0.500		[b] 1.150	27	[b] 0.915	[b] 0.830
10.54	[a] 2.890		[a] 2.815	4.51	[a] 2.615		[a] 2.775	4.13	[a] 2.700	[a] 2.395
27	[b] 0.655		[b] 1.075	41	[b] 0.515		[b] 1.125	29	[b] 0.910	[b] 0.800
10.56	[a] 2.900		[a] 2.770	4.56	[a] 2.610		[a] 2.750	4.15	[a] 2.715	[a] 2.400
29	[b] 0.765		[b] 1.030	46	[b] 0.510		[b] 1.100	31	[b] 0.925	[b] 0.805
11.00	[a] 2.880		[a] 2.770	5.2	[a] 2.600	23.8°	[a] 2.690	4.17	[a] 2.700	[a] 2.390
33	[b] 0.745		[b] 1.030	52	[b] 0.500	C.	[b] 1.040	33	[b] 0.910	[b] 0.795
11.3	[a] 2.845	23.4°	[a] 2.775	5.10	[a] 2.590		[a] 2.675	4.20	[a] 2.670	[a] 2.380
36	[b] 0.700	C.	[b] 1.035	60	[b] 0.510		[b] 1.025	36	[b] 0.880	[b] 0.785
11.14	[a] 2.850		[a] 2.690	5.16	[a] 2.600		[a] 2.675	4.25	[a] 2.645	[a] 2.335
47	[b] 0.705		[b] 0.950	66	[b] 0.500		[b] 1.025	41	[b] 0.845	[b] 0.740
								4.27	[a] 2.625	[a] 2.320
								43	[b] 0.835	[b] 0.725
								4.37	[a] 2.560	[a] 2.190
								53	[b] 0.770	[b] 0.595
	[c] 20 min.		[c] 13 min.		[c] 14 min.		[c] 31 min.		[c] 31 min.	[c] 24 min.

[a] Actual length of axis at time measured.

[b] Change in length of axis after the eye is exposed to 2 c. c. of adrenalin solution for the number of minutes indicated to left in time column.

[c] Time required for each axis to reach a maximum length.

TABLE XVIII. – *Mydriatic action of natural l-adrenalin.*

EXCISED FROG'S EYE.

Experiment 44a. 1 : 125,000.				Experiment 4[illegible]a. 1 : 625,000.				Experiment 47a. 1 : 3,125,000.			
Time.	Long axis.		Short axis.	Time.	Long axis.		Short axis.	Time.	Long axis.		Short axis.
A. m.	*M. m.*		*M. m.*	*A. m.*	*M. m.*		*M. m.*	*A. m.*	*M. m.*		*M. m.*
9.35	*a* 1.810		*a* 1.240	10.09	*a* 2.010		*a* 1.710	10.12	*a* 2.235		*a* 2.055
10.7	*a* 1.745		*a* 1.180	10.11	*a* 2.025		*a* 1.720	11.46	*a* 1.490	23.2°	*a* 0.900
11.13	......		....	10.16	*a* 2.015		*a* 1.690	11.58	*a* 1.670		*a* 1.165
.	....		....	10.42	*a* 1.925		*a* 1.560	*P. m.*			
11.13	*a* 1.690		*a* 1.020	11.26	*a* 1.895		*a* 1.435	12.2	*a* 1.735		*a* 1.290
								1.2	*a* 1.635		*a* 1.080
11.16	In adrenalin.			11.29	In adrenalin.			1.9	In adrenalin.		
11.18	*b* 1.735		*b* 1.175	11.34	*b* 2.075		*b* 1.615	1.12	*b* 1.670		*b* 1.170
2	*c* 0.045		*c* 0.155	5	*c* 0.180		*c* 0.180	3	*c* 0.035		*c* 0.090
11.20	*b* 1.855		*b* 1.265	11.37	*b* 2.135		*b* 1.640	1.15	*b* 1.710		*b* 1.200
4	*c* 0.165		*c* 0.245	8	*c* 0.240	23.0°	*c* 0.205	6	*c* 0.075		*c* 0.120
11.21	*b* 1.880		*b* 1.375	11.40	*b* 2.140	C.	*b* 1.645	1.20	*b* 1.825		*b* 1.400
5	*c* 0.190	22.0°	*c* 0.355	11	*c* 0.245		*c* 0.210	11	*c* 0.190		*c* 0.320
11.26	*b* 1.905	C.	*b* 1.530	*P. m.*				1.26	*b* 1.950	24.2°	*b* 1.645
10	*c* 0.215		*c* 0.510	12.11	*b* 2.195		*b* 1.660	17	*c* 0.315	C.	*c* 0.565
11.29	*b* 2.000		*b* 1.710	42	*c* 0.300		*c* 0.225	1.36	*b* 1.935		*b* 1.610
13	*c* 0.310		*c* 0.690	12.17	*b* 2.195	22.2°	*b* 1.700	27	*c* 0.300		*c* 0.530
11.31	*b* 2.050		*b* 1.775	48	*c* 0.300	C.	*c* 0.265	1.48	*b* 1.850		*b* 1.405
15	*c* 0.360		*c* 0.755	12.41	*b* 2.225		*b* 1.710	39	*c* 0.215	23.2°	*c* 0.325
11.40	*b* 2.240		*b* 1.940	72	*c* 0.330	22.4°	*c* 0.275	2.08	*b* 1.815	C.	*b* 1.310
24	*c* 0.550		*c* 0.920	1.6	*b* 2.310	C.	*b* 1.790	59	*c* 0.180		*c* 0.230
11.42	*b* 2.280		*b* 1.960	97	*c* 0.415		*c* 0.355	2.27	*b* 1.800		*b* 1.370
26	*c* 0.590		*c* 0.940	1.23	*b* 2.340		*b* 1.800	78	*c* 0.165		*c* 0.290
11.44	*b* 2.360		*b* 2.000	114	*c* 0.445		*c* 0.365	2.32	*b* 1.790		*b* 1.430
28	*c* 0.670		*c* 0.980	1.34	*b* 2.325		*b* 1.775	83	*c* 0.155	23.2	*c* 0.350
11.48	*b* 2.430		*b* 2.040	125	*c* 0.430		*c* 0.340	2.37	*b* 1.765	C.	*b* 1.380
32	*c* 0.740	22.0°	*c* 1.020	1.45	*b* 2.335	22.4°	*b* 1.800	88	*c* 0.130		*c* 0.300
11.56	*b* 2.500	C.	*b* 2.105	136	*c* 0.440	C.	*c* 0.365	2.54	*b* 1.780		*b* 1.355
40	*c* 0.810		*c* 1.085	2.12	*b* 2.370		*b* 1.800	105	*c* 0.145		*c* 0.275
11.59	*b* 2.570		*b* 2.190	163	*c* 0.475		*c* 0.365	3.14	*b* 1.790		*b* 1.420
43	*c* 0.880		*c* 1.170	2.30	*b* 2.385		*b* 1.825	125	*c* 0.155		*c* 0.340
P. m.				181	*c* 0.490		*c* 0.390	3.34	*b* 1.760		*b* 1.360
12.1	*b* 2.575		*b* 2.225	2.51	*b* 2.405		*b* 1.830	145	*c* 0.125		*c* 0.280
45	*c* 0.885		*c* 1.205	202	*c* 0.510	22.8°	*c* 0.395	3.54	*b* 1.795		*b* 1.295
12.9	*b* 2.635		*b* 2.345	3.11	*b* 2.420	C.	*b* 1.865	165	*c* 0.160		*c* 0.215
53	*c* 1.045		*c* 1.325	222	*c* 0.525		*c* 0.430	4.31	*b* 1.720		*b* 1.245
12.13	*b* 2.670		*b* 2.395	3.42	*b* 2.445		*b* 1.875	202	*c* 0.085	24.6°	*c* 0.165
57	*c* 1.080	22.2°	*c* 1.375	253	*c* 0.550		*c* 0.440	6.41	*b* 1.645	C.	*b* 1.000
12.21	*b* 2.670	C.	*b* 2.495	4.6	*b* 2.475		*b* 1.855	332	*c* 0.010		*c* 0.080
65	*c* 1.080		*c* 1.475	277	*c* 0.580		*c* 0.420				

[a] Actual length of axis. Measured in 2 c.c. of Ringer solution at [illegible] C.

[b] Actual length of axis at time measured.

[c] Change in length of axis after the eye is exposed to 2 c.c. of adrenalin solution for the number of minutes indicated to left in time column.

TABLE XVIII.—*Mydriatic action of natural l-adrenalin*—Continued.

EXCISED FROG'S EYE—Continued.

Experiment 44a. 1 : 125,000			Experiment 46a. 1 : 625,000.			Experiment 47a. 1 : 3,125,000.		
Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.
11.16	In adrenalin.		11.29	In adrenalin.		1.9	In adrenalin.	
P. m.	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*	*A. m.*	*M. m.*	*M. m.*
12.26	[a]2.670	[a]2.465	4.36	[a]2.485 23.0°	[a]1.825			
70	[b]1.080	[b]1.445	307	[b]0.590 C.	[b]0.390			
12.35	[a]2.645	[a]2.465	4.54	[a]2.480	[a]1.890			
79	[b]1.055	[b]1.445	325	[b]0.585	[b]0.455			
12.44	[a]2.650	[a]2.500	6.51	[a]2.390	[a]1.855			
88	[b]0.960	[b]1.480	442	[b]0.495	[b]0.420			
12.47	[a]2.650	[a]2.500	6.54	[a]2.390	[a]1.870			
91	[b]0.960	[b]1.480	445	[b]0.495 22.4°	[b]0.435			
1.12	[a]2.640	[a]2.525	7.32	[a]2.325 C.	[a]1.840			
116	[b]0.950	[b]1.550	483	[b]0.430	[b]0.405			
1.20	[a]2.655	[a]2.480	7.56	[a]2.255	[a]1.845			
124	[b]0.965	[b]1.460	517	[b]0.360	[b]0.410			
1.35	[a]2.580	[a]2.435						
137	[b]0.890	[b]1.415						
	[c]55 min.	[c]114 min.		[c]307 min.	[c]325 min.		[c]17 min.	[c]145 min.

[a] Actual length of axis at time measured.

[b] Change in length of axis after the eye is exposed to 2 c. c. of adrenalin solution for the number of minutes indicated to left in time column.

[c] Time required for each axis to reach a maximum length.

The readings recorded in Tables XVII and XVIII are taken from eyes immersed in 1:2,000, 1:5,000, 1:25,000, 1:125,000, 1:625,000, and 1:3,125,000 solutions of natural l-adrenalin kept at nearly constant temperatures and lighted by electric light. It will be noticed that with an increased concentration of adrenalin there is a shortening of the time required to cause maximum dilation. If we take the mean of the times in Tables XVII and XVIII required by the long and short axes to reach a maximum length it will be found that—

1:2,000 caused maximum dilation in 16 min.
1:5,000 caused maximum dilation in 22 min.
1:25,000 caused maximum dilation in 70 min.
1:125,000 caused maximum dilation in 84 min.
1:625,000 caused maximum dilation in 316 min.
1:3,125,000 caused maximum dilation in (81)? min.

On the whole the results are not very satisfactory, and if the minimum time necessary for one of the axes be taken to represent the time of dilation the results are even less concordant. The solutions in the order named above cause maximum dilation in 13, 14, 66, 55, 307, and 17 minutes, respectively.

The most reliable results are obtained by comparing two solutions upon eyes from the same frog, thus giving test objects more nearly alike, the one solution being used on the right and the other upon the left eye. Although the eyes from other frogs may show for given sets a longer or shorter "dilation time," as a rule the higher readings will be approximate multiples of the lower, so that the relative values of the coefficients of strengths thus obtained will remain about the same throughout.

As indicated in Table XIX the eyes of frogs Nos. 71, 72, and 73 yield very consistent results. Not only do the dilation times of the weaker solutions agree with each other, but those of the stronger agree and the lengths of these times are roughly proportional to the concentration. At 18° to 19° C.:

1:20,000 adrenalin solution causes maximum dilation in 28 to 29 minutes.
1:40,000 adrenalin solution causes maximum dilation in 27 to 39 minutes.
1:80,000 adrenalin solution causes maximum dilation in 55 minutes.

Table XIX.

	Frog No. 71.			Frog No. 72.			Frog No. 73.		
	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.
	P. m	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*
Right eye in 1:40,000	2.52	3.015	2.425	11.27	3.250	2.550	1.49	3.470	2.985
	2.15	1.916	1.305	10.48	1.950	1.140	1.12	2.160	1.215
	37 min.	1.055	1.120	39 min.	1.300	1.410	37 min.	1.310	1.770
Left eye in 1:20,000	2.45	3.245	2.985	11.46	3.165	2.715			......
	2.17	2.205	1.480	11.17	1.720	0.810			....
	28 min.	1.040	1.505	29 min.	1.445	1.905			....
Left eye in 1:80,000	......		....	.....	...		2.4	3.265	2.810
	....	....			..		1.9	2.175	1.135
							55 min.	1.090	1.675

Weight of frogs, No. 71=30 grams; No. 72=26 grams, No. 73=26 grams.

All the eyes were measured in a Ringer-adrenalin solution kept at 18° to 19° C., lighted by a 16-candlepower incandescent light under a metallic reflector. The maximum dilation is determined by the time required by first axis to reach its greatest length.

RELATIVE MYDRIATIC ACTION OF NATURAL L- AND OF SYNTHETIC DL-ADRENALIN UPON THE EXCISED FROG'S EYE.

A comparison of the degree of mydriasis excited by natural l- and synthetic dl-adrenalin seems to indicate that the former is the more active. This, however, is not brought out very sharply by comparing a large number of eyes promiscuously. For example, if the results of Tables XVII, XVIII, XXI, XXII, XXIII, and XXIV be compared the following relations seem to obtain.

Table XX.—*Comparison of time required to cause maximum mydriasis.*

Concentration of solution	Synthetic dl-.		Natural l-.		Synthetic dl	
	Long axis.	Short axis.	Long axis.	Short axis.	Long axis.	Short axis.
	Min.	*Min.*	*Min.*	*Min.*	*Min.*	*Min.*
1:2,000	21	21	20	13	15	15
1:5,000	23	16	14	31	17	26
1:25,000	56	38	31	24	61	54
1:125,000	153	203	55	114	332	359
1:625,000	74	74	307	325	367	367
1:3,125,000	67	79	17	145	243	275

It will be noticed that one of the axes of the eyes in natural l-adrenalin nearly always reaches a maximum length before those in synthetic dl-adrenalin solution of like concentration. Furthermore, solutions less dilute than 1:125,000 are uncertain in their action, the dilation time often becoming less than that normal for solutions of greater concentration. This is brought out more clearly in Tables XVIII and XXII, in which it will be seen that the dilation time is very irregular for dilute solutions.

Table XXI.—*Mydriatic action of synthetic dl-adrenalin.*

EXCISED FROG'S EYE.

Experiment 50b. 1:2,000.			Experiment 52a. 1:5,000.			Experiment 55a. 1:25,000.		
Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.
P. m.	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*
1.44	[a] 1.720	[a] 1.310	1.46	[a] 1.810	[a] 1.435	2.12	[a] 1.885	[a] 1.815
2.50	[a] 1.690	[a] 1.275	....	....	....	....	....	....
....	....	....	....	....	....	....	....	....
....	....	....	....	....	....	....	....	....
3.1	[a] 1.690	[a] 1.360	1.55	[a] 1.876	[a] 1.585	2.21	[a] 1.980	[a] 1.920
3.3	In adrenalin.		1.57	In adrenalin.		2.23	In adrenalin.	
3.6	[b] 1.745	[b] 1.440	1.59	[b] 1.900	[b] 1.880	2.26	[b] 2.190	[b] 2.125
3	[c] 0.055	[c] 0.080	2	[c] 0.030	[c] 0.295	3	[c] 0.210	[c] 0.205
3.9	[b] 1.885	[b] 1.750	2.2	[b] 2.140	[b] 2.235	2.28	[b] 2.205	[b] 2.095
6	[c] 0.195	[c] 0.390	5	[c] 0.270	[c] 0.650	5	[c] 0.215	[c] 0.175
3.12	[b] 2.095	[b] 2.010	2.4	[b] 2.485	[b] 2.460	2.30	[b] 2.225	[b] 2.135
9	[c] 0.405	[c] 0.655	7	[c] 0.615	[c] 0.875	7	[c] 0.245	[c] 0.215

[a] Actual length of axis. Measured in 2 c. c. of Ringer solution at 23° C.

[b] Actual length of axis at time measured.

[c] Change in length of axis after the eye is exposed to 2 c. c. of adrenalin solution for the number of minutes indicated to left in time column.

TABLE XXI. *Mydriatic action of synthetic dl-adrenalin*—Continued.

EXCISED FROG'S EYE—Continued.

Experiment 50b. 1:2,000.			Experiment 52a. 1:5,000.			Experiment 55a. 1:25,000.		
Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.
3.3	In adrenalin.		1.57	In adrenalin.		2.23	In adrenalin.	
P. m.	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*
3.14	[a] 2.290	[a] 2.220	2.6	[a] 2.510	[a] 2.530	2.35	[a] 2.355	[a] 2.215
11	[b] 0.600	[b] 0.860	9	[b] 0.640	[b] 0.945	12	[b] 0.375	[b] 0.295
3.16	[a] 2.485	[a] 2.390	2.9	[a] 2.695	[a] 2.620	2.37	[a] 2.400	[a] 2.310
13	[b] 0.795	[b] 1.030	12	[b] 0.825	[b] 1.035	14	[b] 0.420	[b] 0.390
3.18	[a] 2.650	[a] 2.600	2.11	[a] 2.775	[a] 2.640	2.39	[a] 2.460	[a] 2.375
15	[b] 0.960	[b] 1.240	14	[b] 0.905	[b] 1.055	16	[b] 0.480	[b] 0.455
3.20	[a] 2.730	[a] 2.680	2.13	[a] 2.875	[a] 2.650	2.42	[a] 2.500	[a] 2.400
17	[b] 1.040	[b] 1.320	16	[b] 1.005	[b] 1.065	19	[b] 0.520	[b] 0.480
3.22	[a] 2.730	[a] 2.760	2.15	[a] 2.885	[a] 2.600	2.45	[a] 2.330	[a] 2.420
19	[b] 1.040	[b] 1.400	18	[b] 1.015	[b] 1.015	22	[b] 0.550	[b] 0.500
3.24	[a] 2.820	[a] 2.800	2.18	[a] 2.960	[a] 2.575	2.47	[a] 2.580	[a] 2.455
21	[b] 1.130	[b] 1.440	21	[b] 1.090	[b] 0.990	24	[b] 0.600	[b] 0.535
3.26	[a] 2.810	[a] 2.775	2.20	[a] 2.970	[a] 2.520	2.50	[a] 2.590	[a] 2.450
23	[b] 1.120	[b] 1.415	23	[b] 1.100	[b] 0.935	27	[b] 0.610	[b] 0.530
3.30	[a] 2.810	[a] 2.750	2.22	[a] 2.955	[a] 2.510	2.52	[a] 2.590	[a] 2.465
27	[b] 1.120	[b] 1.390	25	[b] 1.085	[b] 0.925	29	[b] 0.610	[b] 0.545
3.35	[a] 2.805	[a] 2.760	2.27	[a] 2.960	[a] 2.490	2.54	[a] 2.600	[a] 2.470
32	[b] 1.115	[b] 1.400	30	[b] 1.090	[b] 0.905	31	[b] 0.620	[b] 0.550
3.37	[a] 2.815	[a] 2.725	2.36	[a] 2.955	[a] 2.475	2.57	[a] 2.610	[a] 2.465
34	[b] 1.125	[b] 1.435	39	[b] 1.085	[b] 0.890	34	[b] 0.630	[b] 0.545
3.42	[a] 2.800	[a] 2.725	2.44	[a] 2.945	[a] 2.515	2.59	[a] 2.635	[a] 2.435
39	[b] 1.110	[b] 1.435	47	[b] 1.075	[b] 0.930	36	[b] 0.655	[b] 0.515
3.58	[a] 2.765	[a] 2.620	4.4	[a] 2.850	[a] 2.325	3.1	[a] 2.670	[a] 2.435
55	[b] 1.075	[b] 1.260	127	[b] 0.980	[b] 0.740	38	[b] 0.690	[b] 0.575
4.05	[a] 2.765	[a] 2.610				3.4	[a] 2.675	[a] 2.410
62	[b] 1.075	[b] 1.250				41	[b] 0.695	[b] 0.490
						3.6	[a] 2.710	[a] 2.435
						43	[b] 0.730	[b] 0.515
						3.8	[a] 2.715	[a] 2.410
						45	[b] 0.735	[b] 0.490
						3.10	[a] 2.730	[a] 2.405
						47	[b] 0.750	[b] 0.495
						3.19	[a] 2.740	[a] 2.400
						56	[b] 0.760	[b] 0.480
						3.21	[a] 2.725	[a] 2.270
						58	[b] 0.745	[b] 0.350
						3.23	[a] 2.690	[a] 2.195
						60	[b] 0.710	[b] 0.275
						4.30	[a] 2.485	[a] 2.050
						67	[b] 0.505	[b] 0.130
	[c] 21 min.	[c] 21 min.		[c] 23 min	[c] 16 min.		[illegible]	[c] 38 min.

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[a] Actual length of axis at time measured.

[b] Change in length of axis after the eye is exposed to 2 c. c. of adrenalin solution for the number of minutes indicated to left in time column.

[c] Time required for each axis to reach a maximum length.

TABLE XXII. – *Mydriatic action of synthetic dl-adrenalin.*

EXCISED FROG'S EYE.

Experiment 57a. 1: 125,000.			Experiment 60a. 1:625,000.			Experiment 62a. 1:3,125,000.		
Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.
A. m.	*M m.*	*M. m.*	*A. m.*	*M.m.*	*M. m.*	*A. m.*	*M. m.*	*M. m.*
9.57	[a]2.215	[a]2.020	10.43	[a]1.930	[a]1.870	11.5	[a]2.515	[a]2.550
10.4	[a]2.255	[a]2.035	10.45	[a]1.995	[a]1.915	11.28	[a]2.285	[a]2.190
... ...	[a]2.175	...	11.11	[a]1.950	[a]1.650	11.35	[a]2.240	[a]2.140
10.42	[a]2.175	[a]1.920	...	.....	....	11.55	[a]2.030	[a]1.765
11.29	[a]2.110	[a]1.750	11.53	[a]1.905	[a]1.640	*P. m.*		
						12.32	[a]2.160	[a]1.975
11.31	In adrenalin.		11.55	In adrenalin.		12.37	In adrenalin.	
11.33	[b]2.230	[b]1.900	11.57	[b]2.010	[b]1.745	12.41	[b]2.350	[b]2.160
2	[c]0.120	[c]0.150	2	[c]0.105	[c]0.105	4	[c]0.190	[c]0.185
11.35	[b]2.305	[b]1.915	*P. m.*			12.43	[b]2.410	[b]2.285
4	[c]0.195	[c]0.165	12.3	[b]2.075	[b]1.860	6	[c]0.250	[c]0.310
11.39	[b]2.405	[b]2.045	8	[c]0.170	[c]0.220	12.52	[b]2.520	[b]2.420
8	[c]0.295	[c]0.295	12.10	[b]2.090	[b]1.910	15	[c]0.360	[c]0.445
11.43	[b]2.435	[b]2.180	15	[c]0.185	[c]0.270	12.54	[b]2.535	[b]2.390
12	[c]0.325	[c]0.430	12.25	[b]2.140	[b]1.890	17	[c]0.375	[c]0.415
11.53	[b]2.485	[b]2.240	30	[c]0.235	[c]0.250	1.6	[b]2.495	[b]2.335
22	[c]0.375	[c]0.490	12.29	[b]2.190	[b]1.870	29	[c]0.335	[c]0.360
12.00	[b]2.475	[b]2.245	34	[c]0.285	[c]0.230	1.12	[b]2.490	[b]2.310
29	[c]0.365	[c]0.495	12.40	[b]2.225	[b]1.885	35	[c]0.330	[c]0.335
P. m.			45	[c]0.320	[c]0.245	1.27	[b]2.465	[b]2.280
12.3	[b]2.515	[b]2.235	12.46	[b]2.230	[b]1.920	50	[c]0.305	[c]0.305
32	[c]0.405	[c]0.485	57	[c]0.325	[c]0.280	1.44	[b]2.520	[b]2.335
12.7	[b]2.470	[b]2.185	12.58	[b]2.250	[b]1.870	67	[c]0.360	[c]0.360
36	[c]0.360	[c]0.435	63	[c]0.345	[c]0.230	1.56	[b]2.515	[b]2.375
12.21	[b]2.505	[b]2.175	1.1	[b]2.240	[b]1.935	79	[c]0.355	[c]0.400
50	[c]0.395	[c]0.425	66	[c]0.335	[c]0.295	2.19	[b]2.485	[b]2.240
12.24	[b]2.605	[b]2.215	1.6	[b]2.265	[b]1.990	102	[c]0.325	[c]0.265
53	[c]0.495	[c]0.465	71	[c]0.360	[c]0.350	2.30	[b]2.570 22.8°	[b]2.300
12.30	[b]2.610	[b]2.240	1.9	[b]2.295	[b]2.025	113	[c]0.350 C.	[c]0.325
59	[c]0.500	[c]0.490	74	[c]0.390	[c]0.385	2.39	[b]2.510	[b]2.305
12.34	[b]2.655	[b]2.230	1.18	[b]2.225	[b]1.940	122	[c]0.350	[c]0.330
63	[c]0.545	[c]0.480	83	[c]0.320	[c]0.300	2.56	[b]2.410	[b]2.300
12.36	[b]2.690	[b]2.245	1.23	[b]2.005	[b]1.940	139	[c]0.250	[c]0.325
65	[c]0.580	[c]0.495	88	[c]0.300	[c]0.300	3.18	[b]2.510	[b]2.340
12.40	[b]2.740	[b]2.240	1.39	[b]2.145	[b]1.750	161	[c]0.350	[c]0.365
69	[c]0.630	[c]0.490	108	[c]0.240	[c]0.110	3.31	[b]2.385	[b]2.185
12.50	[b]2.830	[b]2.290	1.48	[b]2.145	[b]1.845	174	[c]0.225	[c]0.210
79	[c]0.720	[c]0.540	113	[c]0.240	[c]0.205	3.45	[b]2.400	[b]2.255
12.54	[b]2.845	[b]2.345				188	[c]0.240	[c]0.280
83	[c]0.735	[c]0.595				4.18	[b]2.445	[b]2.255

[a] Actual length of axis measured in 2 c. c. of Ringer solution at 23° C.

[b] Actual length of axis at time measured.

[c] Change in length of axis after the eye is exposed to 2 c. c. of adrenalin solution for the number of minutes indicated to left in time column.

TABLE XXII.—*Mydriatic action of synthetic dl-adrenalin*—Continued.

EXCISED FROG'S EYE—Continued.

Experiment 57a. 1:125,000.			Experiment 60a. 1:625,000.			Experiment 62a. 1:3,125,000.		
Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.	Time.	Long axis.	Short axis.
11.31	In adrenalin.		11.55	In adrenalin.		12.37	In adrenalin.	
P. m.	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*	*P. m.*	*M. m.*	*M. m.*
1.6	[a]2.850	[a]2.355				221	[b]0.285	[b]0.280
95	[b]0.740	[b]0.605						
1.8	[a]2.860	[a]2.375						
97	[b]0.750	[b]0.625						
1.22	[a]2.950	[a]2.465						
111	[b]0.840	[b]0.715						
1.27	[a]2.945	[a]2.570						
116	[b]0.835	[b]0.760						
1.33	[a]2.925	[a]2.540						
122	[b]0.815	[b]0.790						
1.53	[a]2.930	[a]2.530						
142	[b]0.820	[b]0.780						
2.1	[a]2.970	[a]2.540						
150	[b]0.860	[b]0.790						
2.4	[a]2.980	[a]2.545						
153	[b]0.870	[b]0.795						
2.17	[a]2.905	[a]2.495						
166	[b]0.795	[b]0.745						
22.23	[a]2.950	[a]2.500						
172	[b]0.840	[b]0.750						
2.26	[a]2.965	[a]2.540						
175	[b]0.855	[b]0.790						
2.32	[a]2.970	[a]2.520						
181	[b]0.860	[b]0.770						
2.43	[a]2.920	[a]2.500						
192	[b]0.810	[b]0.750						
2.52	[a]2.935	[a]2.500						
201	[b]0.825	[b]0.800						
2.54	[a]2.945	[a]2.575						
203	[b]0.835	[b]0.825						
2.57	[a]2.955	[a]2.540						
206	[b]0.845	[b]0.790						
3.9	[a]2.795	[a]2.540						
218	[b]0.685	[b]0.790						
	[c]153 min.	[c]203 min.		[c]74 min.	[c]74 min.		[illegible]	[illegible]

[a] Actual length of axis at time measured.

[b] Change in length of axis after the eye is exposed to 2 c. c. of adrenalin solution for the number of minutes indicated to left in this column.

[c] Time required for each axis to reach a maximum length.

TABLE XXIII. —*Mydriatic action of synthetic dl-adrenalin.*

EXCISED FROG'S EYE.

Experiment 39b. 1: 2,000.				Experiment 41b. 1: 5,000.				Experiment 43b. 1: 25,000.			
Time.	Long axis.		Short axis.	Time.	Long axis.		Short axis.	Time.	Long axis.		Short axis.
A. m.	*M. m.*		*M. m.*	*P. m.*	*M. m.*		*M. m.*	*A. m.*	*M. m.*		*M. m.*
10.24	[a]2.165		[a]1.650	2.51	[a]2.000		[a]1.690	10.16	[a]1.950		[a]1.660
......	......		....	.			.		..		......
11.24	[a]1.665		[a]1.260	2.54	[a]2.050		[a]1.735	10.30	[a]1.950		[a]1.225
11.25	In adrenalin			2.55	In adrenalin			10.31	In adrenalin.		
Time in adrenalin solution.	Increase in length of axes.			Time in adrenalin solution.	Increase in length of axes.			Time in adrenalin solution.	Increase in length of axes.		
Minutes	Long axis.		Short axis.	Minutes	Long axis.		Short axis.	Minutes	Long axis.		Short axis.
	M. m.		*M. m.*		*M. m.*		*M. m.*		*M. m.*		*M. m.*
2	[b]0.230		[b]0.140	2	[b]0.105		[b]0.390	3	[b]0.000		[b]0.165
4	[b]0.285		[b]0.315	4	[b]0.400		[b]0.795	5	[b]0.050	21.2°	[b]0.275
6	[b]0.390	23.8°	[b]0.530	6	[b]0.620		[b]0.945	7	[b]0.090	C.	[b]0.490
8	[b]0.650	C.	[b]0.845	8	[b]0.700		[b]1.025	9	[b]0.160		[b]0.490
10	[b]0.885	23.0°	[b]1.115	10	[b]0.785		[b]4.965	11	[b]0.250		[b]1.175
12	[b]1.015	C.	[b]1.280	14	[b]0.975		[b]1.080	13	[b]0.310		[b]0.185
15	[b]1.060		[b]1.325	17	[b]1.010	23.9°	[b]1.080	15	[b]0.385		[b]0.915
18	[b]0.990	23.1°	[b]1.305	20	[b]0.980	C.	[b]1.130	17	[b]0.450		[b]1.045
20	[b]0.950	C.	[b]1.275	22	[b]0.970		[b]1.180	20	[b]0.615		[b]1.175
23	[b]0.935		[b]1.270	24	[b]0.965		[b]1.195	25	[b]0.740		[b]1.310
				26	[b]0.910		[b]1.205	27	[b]0.780		[b]1.330
				30	[b]0.865		[b]1.170	29	[b]0.825	21.2°	[b]1.365
				33	[b]0.815	23.8°	[b]1.140	32	[b]0.890	C.	[b]1.400
						C.		35	[b]0.915		[b]1.455
								37	[b]0.935		[b]1.515
								39	[b]0.970		[b]1.515
								41	[b]0.990		[b]1.555
								43	[b]1.045		[b]1.605
								46	[b]1.050		[b]1.585
								48	[b]1.085		[b]1.610
								50	[b]1.110		[b]1.640
								52	[b]1.125		[b]1.660
								54	[b]1.145		[b]1.685
								56	[b]1.175	21.6°	[b]1.665
								61	[b]1.225	C.	[b]1.685
								66	[b]1.210		[b]1.685
								69	[b]1.205		[b]1.665
	[c]15 min.		[c]15 min.		[c]17 min.		[c]26 min.		[c]61 min.		[c]54 min.

[a] The actual length of the axes are given only for the first and last reading while in Ringer at 23.°

[b] Represent the increase in length of the axis after exposure to adrenalin solution for the number of minutes indicated to the left in the time column.

[c] Time required for each axis to reach a maximum length.

TABLE XXIV.—*Mydriatic action of synthetic [illegible] adrenalin.*

EXCISED FROG'S EYE.

Experiment 44b. 1:125,000.				Experiment 46b. 1:625,000.				Experiment 47b. 1:3,125,000.			
Time.	Long axis.		Short axis.	Time.	Long axis.		Short axis.	Time.	Long axis.		Short axis.
A. m.	*M. m.*		*M. m.*	*A. m.*	*M. m.*		*M. m.*	*A. m.*	*M. m.*		*M. m.*
9.30	[a]1.820		[a]1.230	10.4	[a]1.940		[illegible]	10.4	[a]2.200		[a]2.250
...	...		...								
9.47	[a]1.805	20.8° C	[a]1.165	10.48	[a]1.875		[a]1.475	11.20	[a]1.670		[a]1.125
9.48	In adrenalin.			10.50	In adrenalin.			11.22	In adrenalin.		
Time in adrenalin solution.	Increase in length of axes.			Time in adrenalin solution.	Increase in length of axes.			Time in adrenalin solution.	Increase in length of axes.		
Minutes.	Long axis.		Short axis.	Minutes.	Long axis.		Short axis.	Minutes.	Long axis.		Short axis.
	mm.		*mm.*		*mm.*		*mm.*		*mm.*		*mm.*
4	[b]0.105		[b]0.090	4	[b]0.035		[b]0.105	4	[b]0.015	23.2°	[b]0.000
7	[b]0.085		[b]0.095	6	[b]0.165		[b]0.210	10	[illegible]	C	[illegible]
9	[b]0.045		[b]0.150	14	[illegible]		[illegible]	21	[b]0.040		
11	[b]0.090		[b]0.190	27	[illegible]		[illegible]	32	[illegible]		[illegible]
14	[b]0.090		[b]0.325	31	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]		[illegible]
16	[b]0.130	20.8°	[b]0.395	53	[illegible]	C	[illegible]	49	[b]0.130		[b]0.130
22	[b]0.160	C	[b]0.470	73	[b]0.125		[b]0.070	70	[b]0.185		[b]0.210
25	[b]0.175	21.0°	[b]0.495	77	[b]0.130		[illegible]	[illegible]	[b]0.170		[b]0.115
28	[b]0.270	C	[b]0.560	91	[b]0.140		[illegible]	103	[b]0.200		[b]0.145
30	[b]0.270		[b]0.620	100	[illegible]		[illegible]	115	[illegible]		[illegible]
32	[b]0.320		[b]0.650	113	[b]0.160		[b]0.040	127	[b]0.190		[b]0.115
35	[b]0.350		[b]0.670	133	[b]0.200	22.4°	[b]0.075	130	[b]0.195		[b]0.120
37	[b]0.350		[b]0.700	156	[illegible]	C	[illegible]	149	[b]0.130	22.3°	[b]0.030
41	[b]0.395	21.4°	[b]0.720	160	[illegible]		[illegible]	[illegible]	[illegible]	C	[illegible]
47	[b]0.485	C	[b]0.800	179	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]
52	[b]0.490		[b]0.800	182	[b]0.325	[illegible]	[illegible]	[illegible]	[b]0.130		[b]0.080
59	[b]0.460		[b]0.785	199	[b]0.350	C	[illegible]	192	[b]0.175		[illegible]
62	[b]0.470		[b]0.805	224	[b]0.430		[b]0.150	209	[illegible]		[illegible]
64	[b]0.485		[b]0.815	238	[b]0.425		[b]0.140	234	[b]0.215		[b]0.230
67	[b]0.510		[b]0.830	264	[b]0.495	22.8°	[b]0.190	249	[b]0.280		[b]0.285
72	[b]0.510		[b]0.845	289	[b]0.500	C	[illegible]	275	[b]0.225		[b]0.365
82	[b]0.555		[b]0.875	390	[b]0.535		[b]0.260	312	[b]0.180	24.6°	[b]0.265
95	[b]0.520	22.0°	[b]0.740	344	[b]0.505		[b]0.290	427	[illegible]	C	[illegible]
107	[b]0.565	C	[b]0.805	367	[illegible]		[illegible]				
109	[b]0.580		[b]0.860	477	[illegible]		[illegible]				
123	[b]0.585		[b]0.775	491	[illegible]	[illegible]	[illegible]				
125	[b]0.620		[b]0.795	520	[illegible]	C	[illegible]				
136	[b]0.650		[b]0.785	548	[b]0.175		[b]0.215				
138	[b]0.660		[b]0.825								
161	[b]0.685		[b]0.900								

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[a] The actual length of the axis is given only for [illegible]

[b] Represent the increase in length of the axis after exposure to [illegible] minutes indicated to the left of the time column.

TABLE XXIV.—*Mydriatic action of synthetic and natural adrenalin*—Continued.

EXCISED FROGS' EYES—Continued.

Experiment 46b. 1:125,000.			Experiment [illegible]. 1:625,000.			Experiment 47b. 1:3,125,000.		
9:48	In adrenalin.		11:50	In adrenalin.		[illegible]	In adrenalin.	
Time in adrenalin solution.	Increase in length of axes.		Time in adrenalin solution.	Increase in length of axes.		Time in adrenalin solution.	Increase in length of axes.	
Minutes.	Long axis.	Short axis.	Minutes.	Long axis.	Short axis.	Minutes.	Long axis.	Short axis.
	mm.	*mm.*		*mm.*	*mm.*		*mm.*	*mm.*
164	*a* 0.720	0.9[illegible]						
181	0.69[illegible]	[illegible]						
183	0.70[illegible]	1.0[illegible]						
197	*a* 0.750	1.130						
199	*a* 0.750	*a* 1.140						
219	*a* 0.740	1.180						
222	0.740	1.200						
258	*a* 0.765	*a* 1.2[illegible]						
260	*a* 0.815	*a* 1.275						
281	*a* 0.915	*a* 1.285						
286	*a* 0.9[illegible]	*a* 1.310						
318	*a* 0.980	1.3[illegible]						
325	*a* 0.980	*a* 1.355						
332	*a* 0.980	*a* 1.375						
335	*a* 0.980	*a* 1.375						
359	*a* 0.980 22 [illegible]	*a* 1.355						
362	*a* 0.980 (	*a* 1.355						
388	*a* 0.955	*a* 1.325						
	b 332 min.	*b* 290 min.		*b* [illegible] min.	*b* 357 min.		*b* 215 min.	*b* 275 min.

a Represents the increase in length of the axis after exposure to adrenalin solution for the number of minutes indicated to the left in the time column.

b Time required for each axis to reach its maximum length.

It is difficult to determine the relative mydriatic activity of these two products by simply comparing the results obtained with eyes from different frogs, for when this is done the individual variations are so great that the general average of the dilation times is about the same for like concentrations as is shown by taking an equal number of readings from groups of eyes.

	Synthetic.	Natural.
	Min.	*Min.*
1:1,000 . .	21	1[illegible]
1:2,000 . .	20	20
1:5,000 . . .	24	26
1:25,000 . .	50	51
1:125,000 .	[illegible]2	173

If one were to use these figures to decide, the indication would be that the natural l- and synthetic dl- products have an equal mydriatic action.

As is shown in a study of large numbers of eyes a 1:2,000 solution may in certain cases bring about maximum dilation in as short a time as a 1:1,000 solution, and in rare instances a 1:5,000 solution may compare very favorably with a 1:1,000 solution, and thus throw more or less doubt upon such determinations as agree with results obtained by the blood-pressure method. This criticism holds for eyes of different frogs; but it does not hold when comparing a 1:1,000 solution with a 1:5,000 solution upon the right eye and left eye of the same frog. In this case a series of experiments will quickly determine which is the stronger of the two solutions, and by proper checking against a solution of known strength the approximate strength of the unknown can be determined. All later experiments with the pupil indicate that the relative activity for natural l- and synthetic dl- adrenalin is about the same as determined by blood-pressure experiments. In experiment 75 the right eye was placed in a 1:40,000 solution of natural l- adrenalin, the left in a 1:40,000 solution of synthetic dl- adrenalin. The former dilated to a maximum in twenty-seven minutes; the latter in forty-six minutes. The pupils were then placed in several changes of fresh Ringer, whereupon they gradually constricted. Upon placing them again in fresh 1:40,000 solutions the right eye dilated in the natural l- in sixty-eight minutes and the left eye in synthetic dl- in eighty minutes. This and similar experiments now in progress illustrate in a general way the relative mydriatic action of these two compounds much more truly than is illustrated in a summary of my older experiments.

In conclusion it may be said that a method for the standardizing of adrenalin preparations which involves the idea of minimum doses is unreliable. The pupil method is no exception to this rule, for minimum doses are even more uncertain in their action upon the frog's eye than they are in blood-pressure experiments. It may be assumed that adrenalin acts upon muscle much as does a tetanic stimulus, in which case to secure comparative results upon a given muscle the stimulus, preferably an optimal one, must be of like intensity and duration. In any case the pupil reacts at different rates with different concentration of adrenalin. And the time required for dilation seems to be a better index to the strength of chemical stimulus than the amount of dilation. When the time intervening between the moment of stimulation and that of maximum dilation is chosen as the unit results may be obtained that compare very favorably with those obtained in blood pressure experiments. The synthetic dl- adrenalin has about the same activity as is indicated by the blood-pressure and toxicity experiments already discussed.

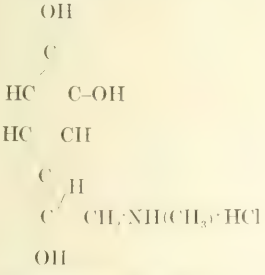
THEORETICAL.

In reading the literature on the active substance of the adrenal gland one is impressed with the difference of the doses said to be lethal and of the amount of substance cited as the minimum dose necessary to cause a rise of blood pressure. These variations, I believe, are to be explained in part by differences of technique but more especially by varying amounts of adrenalin in the preparations used by the different observers. Certainly in experiments antedating 1900 the various workers must have been using at best only mixtures of adrenalin and other substances or compounds of adrenalin itself. Even the epinephrin used by Amberg seems to have had a relatively low degree of toxicity. However, the experiments with epinephrin are worth repeating: perhaps there was some error in determining the lethal dose. If epinephrin proved to have all of the properties of the pure base now known as adrenalin it would be possible to adopt this name for the active substance of the adrenal gland, which from many standpoints is to be preferred above all others. As stated earlier in this paper, Battelli worked with an unusually pure product, and while it is not altogether safe to compare results obtained with animals so widely different as mice and rabbits still it is interesting that the lethal doses given in this paper and those cited by Battelli are very nearly alike, viz., 8 to 10 milligrams per kilogram for subcutaneous injections.

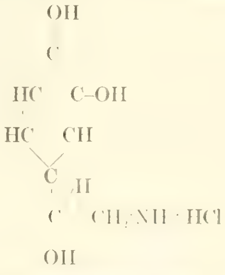
It is also interesting that the relative pharmacological activity of the four compounds, as determined by any given method—say blood pressure, toxicity, or pupil experiments—should bear to each other a rather definite ratio. And when the difference in relative degree of toxicity or of vaso-constrictor activity seems to be closely associated with certain groups in the molecule or to contain arrangements of these groups in space it becomes very suggestive from a theoretical standpoint.

As already pointed out the natural l-adrenalin has a vaso-constrictor action one and a half times as powerful as synthetic dl-adrenalin and nearly the same action as ortho-dioxy-phenyl-ethanol-amin (arterenol), but eighty times as powerful as ortho-ethyl-amino-dioxy-aceto-phenon (homorenon), thus making the relative activity of these substances to each other as the inverse ratio of 1:15:1:80 If, on the other hand, the relative toxicity of the substances jus named be compared it will be found that they are to each other a the inverse ratio of 1:(1.5–2):5:(71–80). In comparing these ratio it will be seen that they are nearly alike, the divergence being i that part of the toxicity ratio pertaining to arterenol. This product though causing nearly the same rises of blood pressure as the natura l-base, is much less toxic. It would seem that the substitution of hydrogen of the amino group by a methyl or ethyl group increases the toxicity; certainly the ethyl group does this, as is shown by the increased toxicity of l- and dl- adrenalin. It would be interesting to test the effect of using different alkyl groups in displacing the hydrogen of the amino group for I am quite certain that if other radicles or more of them were introduced the toxicity would be greatly altered.

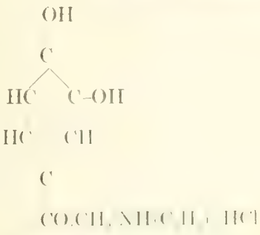
The four compounds, all catechol derivatives, possess pharmacological qualitative properties that are very nearly identical, but their quantitative properties differ widely. What is the key to this quantitative difference? Dakin and others have attributed the similarity in action to the catechol radicle, which is essential to produce a rise of blood pressure. Dakin would divide the adrenalin molecule into two parts, the catechol nucleus and the side chain, oxyethylmethylamine. It was first shown that catechol itself is quite active and that methyl-amino-acetyl-catechol is still more like adrenalin. Furthermore, it was observed that if the hydrogen of the hydroxyl groups were displaced, this compound became inactive. Then, again, the methyl ether of catechol is inactive. Now, in the three compounds—



Ortho-dioxy-phenyl-ethanol-methylamine hydrochloride
(1) Dl-adrenalin hydrochloride



Ortho-dioxy-phenyl-ethanol-amine hydrochloride
(2) Arterenol hydrochloride



Ethyl-amino-aceto-catechol hydrochloride
(3) Homorenon hydrochloride

we have a good illustration of the effects of changing the composition of the side chains. In (1) the composition is the same as (2), with the exception that one hydrogen is displaced by the methyl group while in (3) one hydrogen of the amino group is displaced by an ethyl group and the side chain joined to the catechol nucleus through a carboxyl group instead of a secondary alcohol group. Evidently, it is the nature of this side chain that determines the degree of activity as well as its relation. I agree with Dakin in his contention that the hydrogen atoms of the hydroxyl groups must be unsubstituted, that alkyl groups of low molecular weight tend to increase the activity much more efficiently than when larger molecules are introduced into the side chain. Indeed, several factors seem to be of prime importance in determining the activity of catechol compounds. (1) Whether the substance partakes of the nature of a ketone or of a secondary alcohol, the latter being the more active; (2) the nature of the groups displacing the H of the amino groups, as well as the number of displacements; and (3) the arrangement of the asymmetric carbon atom in space, the laevo arrangement usually being an index of the greater activity. At least this is borne out by my own results and by the results of Cushny and others.

With the theory that these are the determining factors in modifying the vaso-constrictor action of such compounds it is hoped that as a working basis it will aid in a more extended study of other compounds possessing physiological activity.

CONCLUSIONS.

1. The blood-pressure method with dogs under morphine-ether anaesthesia, the vagi cut, and very small doses of curare, is the most accurate pharmacological assay for catechol derivatives.

2. The pupil method as modified by the author is a reliable assay for adrenalin but less delicate and more tedious than the blood-pressure method.

3. Synthetic dl-adrenalin is less active as a vaso-constrictor and as a mydriatic than natural l-adrenalin, the ratio being 2:3.

4. The relative vaso-constrictor activity of the catechol derivatives in the order named, l- and dl-, ortho-dioxyphenylethanolmethylamin, ortho-dioxyphenylethanolamin and ortho-ethylaminodioxyacetophenon are to each other as the inverse ratio of 1:1.5:1:80.

5. The toxicity of the substances in the order named in (4) are to each other as the inverse ratio of 1: (1.5 to 2):5:(71 to 80).

6. The relative physiological activity of these catechol derivatives seems to depend upon the substance partaking of the properties of a secondary alcohol or of a ketone, upon the nature and number of groups displacing the hydrogen of the amino group, and upon the arrangement of the asymmetric carbon in space.

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LIST OF HYGIENIC LABORATORY BULLETINS OF THE PUBLIC HEALTH AND MARINE-HOSPITAL SERVICE.

The Hygienic Laboratory was established in New York, at the Marine Hospital on Staten Island, August, 1887. It was transferred to Washington, with quarters in the Butler Building, June 11, 1891, and a new laboratory building, located in Washington, was authorized by act of Congress, March 3, 1901.

The following *bulletins* [Bulls. Nos. 1-7, 1900 to 1902, Hyg. Lab., U. S. Mar.-Hosp. Serv., Wash.] have been issued:

*No. 1.—Preliminary note on the viability of the *Bacillus pestis*. By M. J. Rosenau.

No. 2.—Formalin disinfection of baggage without apparatus. By M. J. Rosenau.

*No. 3.—Sulphur dioxid as a germicidal agent. By H. D. Geddings.

*No. 4.—Viability of the *Bacillus pestis*. By M. J. Rosenau.

No. 5.—An investigation of a pathogenic microbe *B. typhi murium* Danysz applied to the destruction of rats. By M. J. Rosenau.

*No. 6.—Disinfection against mosquitoes with formaldehyd and sulphur dioxid. By M. J. Rosenau.

No. 7.—Laboratory technique: Ring test for indol, by S. B. Grubbs and Edward Francis; Collodium sacs, by S. B. Grubbs and Edward Francis; Microphotography with simple apparatus, by H. B. Parker.

By act of Congress approved July 1, 1902, the name of the "United States Marine-Hospital Service" was changed to the "Public Health and Marine-Hospital Service of the United States," and three new divisions were added to the Hygienic Laboratory.

Since the change of name of the Service the bulletins of the Hygienic Laboratory have been continued in the same numerical order, as follows:

*No. 8.—Laboratory course in pathology and bacteriology. By M. J. Rosenau. (Revised edition, March, 1904.)

*No. 9.—Presence of tetanus in commercial gelatin. By John F. Anderson.

No. 10.—Report upon the prevalence and geographic distribution of hookworm disease (uncinariasis or anchylostomiasis) in the United States. By Ch. Wardell Stiles.

*No. 11.—An experimental investigation of *Trypanosoma lewisi*. By Edward Francis.

*No. 12.—The bacteriological impurities of vaccine virus; an experimental study. By M. J. Rosenau.

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*No. 22. Chloride of zinc as a deodorant, antiseptic, and germicide. By T. B. McClintic.

*No. 23. Changes in the Pharmacopoeia of the United States of America. Eighth Decennial Revision. By Reid Hunt and Murray Galt Motter.

No. 24. The International Code of Zoological Nomenclature as applied to medicine. By Ch. Wardell Stiles.

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TREASURY DEPARTMENT

Public Health and Marine-Hospital Service of the United States

WALTER WYMAN, Surgeon-General

HYGIENIC LABORATORY.—BULLETIN No. 56

M. J. ROSENAU, Director

MARCH, 1909

MILK AND ITS RELATION TO THE PUBLIC HEALTH

[Revised and enlarged edition of Bulletin No. 41]

(BY VARIOUS AUTHORS)

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1. INTRODUCTION.

Milk and its Relation to the Public Health.

INTRODUCTION.

By Walter Wyman,

Surgeon-General, Public Health and Marine-Hospital Service.

During the last few years increasing attention has been given to
k in its relation to the public health. This is especially true in
United States, where the more progressive health authorities of
larger cities and many of the States have been instrumental in
rkedly improving their milk supplies.

The question of sanitary milk is to the American people especially
tinent. Milk is perhaps used to a greater extent in this than in
y other country. It holds a peculiar place in the nation's dietary
ause of its varied applicability. Containing as it does all the
entials of a perfect ration, proteids, carbohydrates, fats, inorganic
ts, and water, it is capable of almost universal use. Because of
s, and, in addition, its facility of ingestion and comparative ease
digestion, it constitutes an important food for the sick and
nvalescent.

Of even greater importance is the use of cow's milk as a substitute
r mother's milk in infant feeding. It will be perceived that those
st dependent upon this food—the sick and convalescent, infants
d children—constitute that part of the community suffering the
eatest injury from the use of a food impaired in its nutritive con-
nt. This is due to the fact that they are least able to resist the
rmful effects of foods contaminated by toxins or pathogenic micro
ganisms. While improved conditions of living have contributed
a steady decrease of the general mortality in civilized countries,
is unfortunately does not apply to the infant population under one
ar of age. It is recognized that gastro-intestinal disease is the
rgest single factor determining infant mortality, a condition in
eat measure due to improper methods of feeding. This enormous
ss of potential wealth is of grave concern to the State and worthy
f most careful consideration. It is especially for these reasons that
e question of sanitary milk and its relation to the public health
allenges our best endeavors.

The investigation into the origin and prevalence of typhoid fever
the District of Columbia during 1906 by a board of medical officers
f the Public Health and Marine-Hospital Service brought out many
cts evidencing the possible danger of milk as a carrier of this di
se, and stimulated investigation and renewed activity in the efforts
secure pure milk supply in the District of Columbia.

This investigation and the work of the Department of Agricultur
concerning the milk supply at the farms were referred to in a lette
to the President, June 11, 1907, by Dr. G. Lloyd Magruder, of Wash
ington, in which it was suggested that the Bureau of Public Healt
and Marine-Hospital Service be directed to make an investigation o
the milk industry in the District of Columbia from the farm to th
consumer, with the cooperation of other departments.[a]

The Surgeon-General, being called upon by the President for a
opinion as to whether such an investigation should be made, replie
affirmatively, with detailed reasons therefor, and with special recom
mendations as to cooperation of the several bureaus in the Depart
ment of Agriculture, and the President and the Secretary of th
Treasury thereupon directed the said investigation.

In order to properly study the subject as it exists in the Dist
of Columbia, it was deemed necessary to treat the matter from
broad point of view; that, to study the local aspect of a world-wid
problem, the findings and experiences of others must necessarily b
considered. In many respects the Federal Government has peculi
advantages for the study of these problems which, strictly speakin
are not confined to any one locality, but are national in scope. It i
therefore incumbent on the National Government to assume its r
sponsibilities and attempt the solution of scientific questions of thi
character influencing the lives and health of its citizens. Because
the relation the Public Health and Marine-Hospital Service bears t
the conservation of the public health it was determined to make th
investigation of such a character that, in addition to being of loc
value, it would also be of assistance to health officers at large, an
especially to those not as yet provided with the necessary laborator
facilities and corps of workers such as can be afforded only by t
richer and more densely populated centers.

It has been the object to include in this volume all available dat
showing the influence of milk as a carrier of infection, its chemic
composition, the contaminations found therein, their influence upo
it as an article of food, and the measures necessary in its produ
tion and handling to prevent such contamination.

Milk in the udder of a healthy cow is rarely sterile, but wit
proper methods can occasionally be removed in small quantities fr
from micro-organisms. In this condition it may theoretically
considered normal milk, and as such has been kept for over two year
But this is not the milk of commerce. In the healthy cow, milk ma
contain organisms while still in the udder, or receive its initial co
tamination with the omnipresent microphyte in its passage throug
the ducts of the animal's teats. This may be considered its first poi
of contact with the outer world, for these organisms in the healt
animal have gained access to the ducts from without. At every oth

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[a] Annual Report, Public Health and Marine-Hospital Service, 1907, p. 35.

oint of contact on its twelve to forty-eight hour journey to the con mer it receives additional bacteria.

Milk holds a peculiar position among foodstuffs in that it is an xcellent medium for the growth of many micro-organisms, both the rdinary saprophytic varieties and those pathogenic to man. These ctors often produce in market milk an enormous bacterial content. akharbekoff found that in St. Petersburg examination of samples of ilk as delivered to the houses showed the presence of from 10,200,000 82,300,000 bacteria per cubic centimeter. Samples of market milk Giessen have shown over 169,000,000 per cubic centimeter. New ork City milk as high as 35,200,000, London milk 31,888,000. In ashington, examinations made at the Hygienic Laboratory of the blic Health and Marine Hospital Service during the summer of 6 showed a maximum of 307,800,000 and an average bacterial ntent of 22,134,289. Were milk transparent, this luxuriant growth ould be evident to the naked eye, but because of its opacity such ntamination occurs unnoticed. Fortunately, most of these organ ms are saprophytes, but there are good reasons to believe that they ay elaborate toxins, rendering milk dangerous as a food.

It is evident, from a broad view of the subject, that a pure and holesome milk supply is possible, and this volume contains all the ecessary information to attain that end, as well as the existing stand ds of purity to which it should conform.

The three cardinal requirements, *cleanliness*, *cold*, and *speedy trans ortation* from the cow to the consumer must be observed, and the cow erself must be free from disease. For their observance, intelligence nd care on the part of the dairyman and milk dealer are absolutely ssential.

The bearing of all these points upon the wholesomeness of milk, s treatment when contaminated, and its use as an article of food, specially for infants, has been treated in detail by the various col aborators. To ascertain how serious an indictment might be returned gainst milk as a carrier of disease, a compilation of epidemics pro duced by this means has been made by Doctor Trask. Reports of 00 epidemics have been abstracted in tabular form and appear in he text. These are only the few that have been reported and are ccessible in the literature; how small a fraction of all cases this must e can only be surmised.

As a result of large experience, Doctor Lumsden describes how the ilk supply of cities becomes contaminated with typhoid bacilli and he best epidemiological methods of determining the influence of milk s a factor in the propagation of typhoid fever.

With a view to determining the presence or absence of tubercle bacilli in the market milk of Washington, Doctor Anderson [illegible]

272 samples from 101 dairies. He found that 6.72 per cent of the samples contained tubercle bacilli virulent for guinea pigs, and that 11 per cent of the dairies whose milk was examined supplied milk containing these micro-organisms in sufficient number and virulence to render guinea pigs tuberculous. The milk purchased by one charitable institution for the use of children caused tuberculosis in the animals upon which it was tested.

Evidence of this character again emphasizes the necessity of applying the tuberculin test among dairy herds, and taking necessary precautions with respect to milk of doubtful character.

In a second paper Doctor Anderson summarizes the evidence proving that Malta fever may be spread by infected goat's milk.

A peculiar disease, known as "milk sickness," is described by Doctor McCoy. Although fortunately rare at the present time, cases continue to occur in the mountainous sections of Tennessee and elsewhere.

Doctor Stiles shows that so far as the zoo-parasitic diseases of man are concerned, there is little to fear concerning the presence of such parasites in milk.

Statistical studies of mortality and morbidity, as influenced by milk, have been made by Doctor Eager. He gives figures to prove that the high infantile mortality may be attributed almost entirely to impure milk.

Doctor Wiley discusses the subject of ice cream, its use as an article of food, its composition, the extent to which it may be contaminated or adulterated, and the result of such contamination upon the public health. He also refers to the established standards governing its manufacture, and presents evidence to show their reasonableness both to the manufacturer and consumer.

Doctors Kastle and Roberts give a general survey of our present knowledge regarding the physical and chemical characteristics of milk, as well as the chemical changes in milk brought about by the action of heat and acids; and also those changes accomplished by the action of enzymes and micro-organisms. The subject of milk adulteration is also considered. It has been shown, as the result of original investigations, that the milk ferments can withstand a temperature of 60° to 65° C. for some time without material injury. Twelve per cent of the samples of Washington market milk examined were found to be below the legal standard, 3.7 per cent gave evidence of having been watered, and a very large proportion of the samples examined contained appreciable quantities of dirt. None of the samples examined contained artificial coloring matters, and only one contained milk preservatives.

Doctor Rosenau shows, as a result of many hundred bacteriologic examinations of the market milk of Washington made in the Hygienic Laboratory, that for the most part it is old, warm and dirty. In the summer of 1906 the market milk contained on an average of 22,134,289 bacteria per cubic centimeter, and was delivered at an average temperature of 16.5° C. During 1907 the average was 11,000,000 bacteria per cubic centimeter, and temperature 14.2° C. The advantages of bacterial counts to the health officer and to the practical dairyman are pointed out.

As a result of original investigations, Doctor Rosenau and Doctor McCoy demonstrate the causes of the phenomenon known as the "germicidal property of milk." They show that the decrease in the number of bacteria in fresh milk is for the most part apparent, not real, and further that the restraining action of milk can not take the place of cleanliness and ice, but may be taken advantage of in good dairy methods.

Doctor Miller reviews the significance of leucocytes and streptococci in milk and points out the unsatisfactory state of our knowledge concerning their sanitary significance.

Doctor Mohler points out that probably the most important disease of cows from the standpoint of public health is tuberculosis, and that it is also the most prevalent. The German commission on tuberculosis found over 10 per cent (6 out of 56) cultures of tubercle bacilli of human origin, virulent for cattle. In a similar series of tests conducted by the British Royal Commission on tuberculosis, 60 cases of the disease in the human being were tested with the result that 14 were claimed by this commission to have been infected from bovine sources. It has been found by Schroeder in this country that even when tubercle bacilli are not being excreted by the udder the dirt and manure of the stables where the diseased animals are kept are in many cases contaminated with tubercle bacilli. This contaminated material may readily infect the milk even though it comes from a healthy cow. In a recent examination at the Bureau of Animal Industry, Experiment Station, of the manure passed by 12 cows purchased from dairy farms in this city and infected with tuberculosis to an extent only demonstrable by the tuberculin test, tubercle bacilli were found in over 41 per cent of the cases.

Mohler estimates that probably 25 per cent of all the cows which supply milk to the District of Columbia are tuberculous. He further points out the great practical value of the tuberculin test and insists that all milk should come from either tuberculin tested cattle or be subjected to pasteurization under the supervision of the Health Department in case the herd is not tuberculin tested.

Mr. Webster, among other things, emphasizes the value of the score card in the sanitary inspection of dairies and its bearing on the production of clean milk. He also gives 21 very useful suggestions concerning the cows, stables, milk houses, and methods of milking and handling milk.

Doctor Bolton writes of the dangers from contaminated water supplies on dairy farms and shows that a pure water supply on the farm appears to present much fewer difficulties than the same problem in towns. Each supply presents its own problem which must be solved for itself, with proper recognition of the objects to be aimed at, and these are purity, abundance, and convenience.

Doctor Bolton also gives the methods and results of the examination of the water supply of dairies supplying the District of Columbia. The analysis of results seems to show that there are comparatively few water supplies on the dairy farms visited which are free from sanitary objection, but in spite of this fact it is nevertheless probable that in many or most cases the faults can be rectified with little expense.

Doctor Melvin offers a practical solution of the classification of market milk. He proposes three grades: (1) Certified milk; (2) inspected milk, and (3) pasteurized milk.

Doctor Kerr gives a brief outline of the organization and conduct of medical milk commissions in the United States, established to foster the production of "certified milk." Emphasis is laid on the fact that the plan was formulated by a physician, and that it contemplates the sanitary supervision of dairies by a commission appointed by the local medical society for the purpose of producing pure milk especially for the use of infants and invalids. In this paper are included copies of the first contract entered into between a medical milk commission and a dairyman; also the requirements of the milk commission of the medical society of the county of New York, which contain all of the essential rules required by other commissions for the production of pure milk.

It appears that this movement has been a potent factor in improving the character of the milk supply in various parts of the country, as it has required that only tuberculosis-free cattle should be used for the production of milk, that their milk should be cooled to a temperature of 45° F. and transported in a manner so that it reaches the consumer before noticeable biological or chemical changes have occurred therein. He also refers to the founding of infants' milk depots in the United States, and presents in tabular form the number of such organizations and other pertinent information relating thereto.

The important subject of pasteurization has been carefully studied by Doctor Rosenau, who points out its advantages and discusses its inconveniences. He recommends 60° C. for twenty minutes as the best temperature to use in pasteurizing milk, as this degree of heat is sufficient to destroy the pathogenic micro-organisms without devitalizing the milk itself. While pasteurization is not the ideal to be sought, practically, it is forced upon us by present conditions. It prevents much sickness and saves many lives—facts which justify its use under proper conditions. It is recommended that in large communities at least, pasteurization should be under direct supervision of the health authorities.

The trend of our modern knowledge upon the important subject of infant feeding is stated in Doctor Schereschewsky's article on this subject. The importance of breast feeding is emphasized. It is shown that the caloric needs of the infant must be considered in order to insure success in artificial feeding. Some of the errors of formula feeding are pointed out, and stress is properly laid upon the disastrous results which frequently ensue from overfeeding, especially with excessive amounts of butter fat. Schereschewsky believes that there is no relation between the heating of milk and infantile scurvy, and shows how this disease may result from qualities in the milk, other than those resulting from heating.

In the last three articles named, as well as elsewhere in this bulletin, references will be observed to the achievements of Mr. Nathan Straus in promoting the use of clean pasteurized milk for infants and the establishment of infants' milk depots both in the United States and abroad, and it is proper here to give recognition to his philanthropic and successful efforts.

Doctor Woodward describes the municipal regulation of the milk supply of the District of Columbia. He recounts the history of the development of the milk inspection service, which consists of supervision, inspection of dairies and dairy farms, and inspection of the milk. It is shown that these measures have resulted in the improvement of the milk supply, and that there has been a notable reduction of morbidity following their inauguration.

The laws and ordinances governing the supervision of milk are given, and in addition copies of the forms of reports, etc., which are of value to those having supervision of milk supplies.

Acknowledgments are here made to Doctor Woodward and the officers of the Bureaus of Animal Industry and Chemistry for their hearty cooperation and contributions upon this important subject.

A SECOND EDITION.

The first edition of this bulletin, which was issued January, 1908, has been of great value to health officers and others interested in improved milk supplies, as is shown by the enormous and constant demand throughout the world for copies. It was abstracted by Mr. Nathan Straus, and the abstracts were generously distributed by him throughout Europe in connection with his propaganda for safe milk.

The first edition has long since been exhausted, and it therefore becomes necessary to publish a second edition. On account of the short time since the first edition appeared and the character of some of the data relating to the investigation, especially that contained in the statistical tables, it has been impracticable to include the corresponding statistics for the year 1908. The limitations of the volume have prevented the inclusion of chapters relating to certain milk products, although discussion dealing with butter, dried milk, prepared milk, and milk substitutes would be of value. It is expected that these subjects will be given consideration in later publications.

Important chapters added in this edition include a discussion of the relationship of the tuberculous cow to public health, by Dr. E. C. Schroeder, of the Bureau of Animal Industry. In this article, Doctor Schroeder invites attention to the manner in which tubercle bacilli are expelled by tuberculous cattle, the technique used in demonstrating bovine tubercle bacilli, the ways in which tubercle bacilli expelled by tuberculous cows get into milk, and the dangers involved therein.

In a chapter on the thermal death point of pathogenic micro-organisms in milk, Surg. M. J. Rosenau concludes that the heating of milk to 60° C. for twenty minutes is sufficient to destroy the tubercle bacillus, the diphtheria bacillus, the cholera vibrio, the dysentery bacillus, and the *Micrococcus melitensis*. He also refers to recently enacted laws relating to pasteurization, discusses home pasteurization, and gives directions for its employment.

Passed Asst. Surg. John F. Anderson, in a new chapter, deals with the relative proportion of bacteria in top milk and bottom milk, and its bearing on infant feeding.

Dr. H. W. Wiley, Chief of the Bureau of Chemistry of the Department of Agriculture, has also contributetd an additional chapter on the national inspection of milk.

It is a pleasure to acknowledge the interest shown in this publication, as well as again express appreciation to Doctor Woodward and the officers of the Department of Agriculture for their cooperation in the preparation of this work.

2. MILK AS A CAUSE OF EPIDEMICS OF TYPHOID FEVER, SCARLET FEVER, AND DIPHTHERIA.

2. MILK AS A CAUSE OF EPIDEMICS OF TYPHOID FEVER, SCARLET FEVER, AND DIPHTHERIA.

By John W. Trask,

Passed Assistant Surgeon, Public Health and Marine-Hospital Service.

That milk may play a part in the spread of certain diseases has, for many years, been appreciated. From our present knowledge the more important of these are typhoid fever, scarlet fever, diphtheria, and possibly tuberculosis.

Milk, from the time it leaves the cow's udder, receives from its surroundings bacteria of various kinds. Certain of these organisms come from the teats of the cow and the dust and dirt of the stable, and are possibly in most cases harmless; others come from the hands of the milker and those handling the milk, and from the pails and cans used for milking, storage, and transportation. During the last fifty years there has been piling up a mass of evidence which would seem to show that milk may receive from man the specific organisms of certain infectious diseases, and that these organisms may retain their virulence for some time and produce the disease in susceptible individuals drinking the raw milk. Many epidemics supposedly spread in this way have been reported in the literature since 1857. Compilations of these cases have been made by Hart [a] in England, Schlegtendal [b] in Germany, Carøe [c] in Denmark, and by Busey [d] and Kober,[e] R. G. Freeman [f] and H. B. Baker [g] in this country.

Up to 1895 Hart, and Busey and Kober had collected 240 such epidemics. In addition to these, there are here presented 260 compiled from the literature and from special reports. (I desire here to ackowledge the great assistance rendered by the many health officers and other physicians who so kindly responded to the circular letter sent out by the Surgeon-General requesting reports of milk epidemics.) The 90 epidemics compiled by Carøe have not been included because of lack of time and space. No attempt has been made to note every outbreak reported as spread by milk; many cases where the evidence did not seem entirely convincing have been omitted. Necessarily much of the evidence upon which it is determined whether or not an epidemic is conveyed by milk is circumstantial; the same may be said of water-borne disease, and indeed of many of the things in daily life which we firmly believe. In an explosive outbreak of an infectious disease, to find that all persons attacked had used one milk supply, that they had apparently nothing else in common, that no cases occurred except among users of this milk, and then to isolate from the milk the specific organism of the disease in a virulent state, is believed to be good evidence in the absence of other explanation. It is not to be inferred that this has been taken as an absolute standard up to which all epidemics must come before being considered as spread by milk, for to do this the outbreak would have to occur in a locality previously entirely free from the disease and the development of secondary-contact cases, which is necessarily a common occurrence, would wrongly exclude such epidemics. Then, too, the difficulty of isolating the Eberth bacillus when in small amount and accompanied by large numbers of other organisms and our lack of absolute knowledge as to its specificity, and the fact that no organism has as yet been isolated which is commonly accepted as the causal agent of scarlet fever, would lead to erroneous conclusions if the isolation of a specific organism were insisted upon.

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[a] Hart (E.), Transactions Internat. Med. Cong. London, 1881, IV, 491, also Brit. Med. Jour. Lond., 1897, 1, 1167, 1229, and 1292.

[b] Schlegtendal, Deut. Vierteljahrschr. f. Offentl. Gesundheitspflege, 1900, Bd. XXXII, 287.

[c] Carøe (K.), Ugeskrift for Laeger, Kobenhavn, 1898, 5 R., V, p. 1009.

[d] Busey (S. C.) and Kober (G. M.), Report of Health Officer of District of Columbia, 1895, p. 299.

[e] Kober (G. M.), Senate Doc. 441, Fifty-seventh Congress, 1st session.

[f] Freeman (R. G.), Medical Record, N. Y., 1896, XLIX, 433.

[g] Baker (H. B.), Annual Report Michigan State Board of Health, 1896.

TYPHOID FEVER.

Schüder[a] in 1901 collected from the literature 650 typhoid epidemics the supposed cause of which had been reported. Four hundred and sixty-two were reported as spread by water, 110 by milk, and 78 by all other means. This places milk second only to water as a carrier of typhoid infection. But the ratio of 462 to 110 probably by no means shows the true relation of water and milk as producers of such outbreaks. Schüder's epidemics were collected mainly from continental Europe, where milk epidemics are apparently not as common as in England and America, due possibly to the more or less customary practice in Europe of using pasteurized or cooked milk. The result of such a compilation as the above may also have been affected by the fact that until comparatively recently water has received much more attention in typhoid epidemiological work than has milk.

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[a] Schüder, Zeitschrift f. Hyg. und Infectionskrankheiten, 1901, XXXVIII, p. 343.

It is evident that Schüder did not include in his list the approximately 90 typhoid epidemics collected by Carøe.[a] These occurred in Denmark between 1878 and 1896, and were reported as in all probability due to milk. It is also apparent that he did not include the combined milk typhoid epidemics collected by Hart, and Busey and Kober, 138 in number, which had been previously compiled.

Undoubtedly the relative importance of the various agencies by which typhoid fever is distributed varies with the locality and conditions. The various factors, water, milk, flies, and contact, have different values in the city and in the town. They will naturally also vary in importance with the season, the latitude, and the local customs. Improved water supplies have eliminated water as a factor in many places, while regulation of the production, handling, and sale of milk is lessening its influence for harm in some communities. It would seem that water has been so apparent as a frequent carrier of the infection that other agents have not been looked for, or at least not commonly found, until improved water supplies had demonstrated that there were other factors at work. The experience in Massachusetts has been given by Harrington[b] as follows:

> In the public mind outbreaks and epidemics of this disease (typhoid) are commonly associated with polluted drinking water, but when water supplies are properly guarded, as in Massachusetts, for example, they are more commonly found to be caused by contaminated food, and especially by that one which is most subject to pollution and which offers the specific organism the most favorable conditions for preserving its virulence and increasing its numbers—namely, milk. During the past two years, of 18 local outbreaks of typhoid fever in different parts of Massachusetts investigated under my direction, 14 were traced to milk.

Jensen[c] also makes the statement:

> The principal means by which typhoid fever is distributed in places where there is a safe and hygienic water supply is through milk.

BACILLUS TYPHOSUS IN MILK.

V. C. Vaughan[d] reported in 1890 the isolation of a bacillus from the water of a dairy well, and from the milk sold by the dairy. There had been one or more cases of typhoid in the family of the milkman, and one or more cases existed in every family patronizing this dairy.[e] The bacillus was highly pathogenic to white rats and guinea pigs. It was nonliquefying and toxicogenic. The bacillus resembled but was not identical with that of Eberth. When the use of the milk was discontinued the outbreak ceased.

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[a] Carøe (K.), Ugeskrift for Laeger, 1898, V, p. 1009.

[b] New York Med. Jour., 1907, p. 697.

[c] Essentials of Milk Hygiene, English ed., 1907, p. 106.

[d] Vaughan (V. C.), Ann. Report State Board of Health, Michigan, 1891, p. 246.

[e] Vaughan (V. C.), Trans. Seventh Internat. Cong. of Hyg. & Demography, 1891, Vol. III, Section III, p. 121.

Dr. A. R. Reynolds,[a] then commissioner of health of Chicago, stated in 1902 that although special search had been frequently made during the last eight years the typhoid bacillus had been found in Chicago city milk only three times, and then in cases of local epidemics, and that in 1902 the presence of the typho-colon group of bacilli had been repeatedly demonstrated.

Konradi[b] isolated the typhoid bacillus from milk in 1905. In Kolozsvar there was an unusual number of cases of typhoid. (See Table of epidemics.) The water could in no way be connected with the increase, and attention was attracted to a bake shop from which many cases seemed to originate. The typhoid bacillus was isolated from a sample of milk taken from this bake shop. Proper precautions were immediately taken against this shop and its milk, and the number of cases of typhoid fell in the next month back to the usual average number. He also examined 32 other samples of milk and isolated the typhoid bacillus from one taken from a dairy where the farmer's son had a mild attack of typhoid fever which was not severe enough to keep him from working and milking the cows.

Shoemaker[c] reports an outbreak of milk typhoid in Philadelphia in October, 1906. He states:

> A culture made from the milk proved the presence of the typhoid bacillus in it.

A visit to the dairy revealed that the proprietor and one of his servants were ill with typhoid and that—

> the son was convalescing from typhoid fever and was filling the milk bottles from a tank by siphonage, starting the flow by sucking with the mouth at one end of the tube. A culture made from this end of the tube revealed many typhoid bacilli.

Cautley[d] infected milk with the typhoid bacillus and recovered the bacillus after seven days. In his summary he states:

> The typhoid bacillus will live in milk under the conditions that ordinarily prevail in a household. When this bacillus has been artficially added in large amount to milk in the condition in which it commonly reaches the consumer, the presence of the microbe in the living state may be demonstrated after the milk thus treated has been kept several days. * * * It will also live in milk which has turned sour at the temperature of the room in which it is kept.

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[a] Reynolds (A. R.), Chicago Medical Recorder, 1902, p. 222.

[b] Konradi, Centralbl. f. Bakt. etc., 1 Abt., Bd. 40, p. 31.

[c] Journal Am. Med. Assn., May 25, 1907, p. 1748.

[d] Cautley (Edmund), Report Med. Officer, Local Govt. Board, London, 1896-97, p. 243.

Broers[a] demonstrated the ability of the typhoid bacillus to live in milk and butter for from two to three weeks.

Bruck in 1903[b] took ordinary market milk and infected it with the bacillus typhosus. He then ran the milk thus treated through a separator and found the viable organism persisting in the cream for ten days after separation. Butter made from this cream showed the presence of the viable bacillus for twenty-seven days. The bacillus typhosus could be recovered from the buttermilk for ten days. Pfuhl[c] showed the ability of the Eberth bacillus to persist in market milk for thirteen days and in butter for twenty-four days.

Eyre[d] undertook experiments to demonstrate the growth of the typhoid bacillus in milk. To avoid the false ideas arising from the use of the sterilized product, he drew the milk from a healthy cow under aseptic conditions and gives the following results showing the possible rate of increase:

	0 hours.	2 hours.	4 hours.	6 hours.	8 hours.	12 hours.	24 hours.
B. typhosus	78	50	12	42	46	460	6,000

This shows a decrease for the first few hours, due to the germicidal action of fresh milk. In another case the count showed the following:

	0 hours.	24 hours.	48 hours.	7 days.
B. typhosus	78	60,000	10,300,000	440,000,000

SUMMARY OF EPIDEMICS.

Of the 179 typhoid epidemics reported as spread by milk, compiled by the writer, 107 occurred in the United States, 43 in Great Britain, 23 in continental Europe, 3 in Australia, 1 in New Zealand, and 2 in Canada; all cases enumerated in the outbreak were reported as living in houses supplied with the suspected milk in 96 of the epidemics; a case, suffering from the disease at such a time as to have been the possible source of infection, was found at the producing farm, distributing dairy, or milk shop in 113 cases; the outbreak was supposed to have been due to bottles returned from infected households and refilled and distributed without previous sterilization in 4 cases; the diseased person or persons were mentioned as handling the milk or milk utensils in 2; the sick milked the cows in 6; the same person nursed the sick and handled the milk or milk utensils in 6; same person was mentioned as nursing sick and milking cows in 10; ice cream was given as the infective medium in 3; whipped cream in 1; typhoid dejecta were reported as thrown on the ground in such a way as to have more than probably contaminated the well water used for washing the milk utensils in 4; in many cases mention was made of special incidence of the disease among persons in the habit of drinking milk; the Eberth bacillus was isolated from the milk in 1 case (Konradi); it was reported that measures taken upon the presumption that milk was the cause of the epidemic, and looking to the removal of this as a factor, were followed by abatement of the outbreak after due allowance for the usual period of incubation from the distribution of the last infected milk in 78 of the cases.

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[a] Broers (C. W.), Nederlandsch. Tijdschrift voor Geneeskunde, 1904, XL, p. 1260.

[b] Bruck, Deut. Med. Woch., 1903, XXIX, p. 460.

[c] Pfuhl, Zeit. Hyg., 1902, XL, p. 555.

[d] Eyre (J. W.), Jour. State Med., London, 1904, XII, p. 728.

The following is an example of a typhoid epidemic apparently due to milk:

STAMFORD EPIDEMIC, APRIL 15 TO MAY 28, 1895.[a]

Stamford, Conn., a town of 15,000 population, had for some months been comparatively free from typhoid fever. During the nine days following April 14, 1895, 160 cases were reported in addition to 24 noted as suspicious. One hundred and forty-seven out of the 160 and all of the suspected cases had used milk delivered by one dairyman, B. Between April 15 and May 28, 386 cases living in 160 houses were reported. The dairy was closed April 21, and on May 6, just fifteen days after the sale of milk was stopped, the outbreak had practically subsided. (See charts 1, 2, and 3.)

Of the 386 cases 352 (91.2 per cent) lived in houses taking milk from dealer B., 12 were known to have used this milk at a café supplied by him, 2 obtained it at a bake shop selling the same milk, and 2 obtained it in other ways, making 368 cases so traced or 95.3 per cent. (See diagram I.) Eight cases were supplied directly by a producer, E. B. L., who produced the bulk of the milk peddled by B. This makes 376, or 97.1 per cent, connected with this milk supply. Of the other cases 4 were supplied by one dealer, 5 were supplied by 5 different dealers, and 1 could not be connected with any milk supply. It was estimated that 3,000 quarts of milk were peddled daily in Stamford, of which B. supplied about 275 quarts. He therefore supplied about one-eleventh of the milk and had 95.3 per cent of

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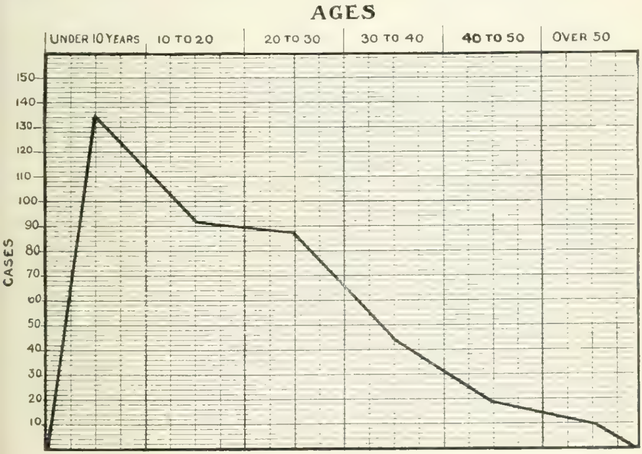
[a] Smith (Herbert E.), Connecticut State Board of Health Report, 1895, pp. 161–179.

CHART 1.

SHOWING IN TEN-YEAR PERIODS THE AGES OF CASES DURING THE STAMFORD OUTBREAK.



NOTE THE UNUSUAL NUMBER OF CASES UNDER 10 YEARS OF AGE AS COMPARED WITH THOSE BETWEEN 20 AND 30 YEARS, THE PERIOD USUALLY MOST SUSCEPTIBLE TO TYPHOID.

CHART 2.

SHOWING IN FIVE-YEAR PERIODS THE AGES OF CASES DURING STAMFORD OUTBREAK.

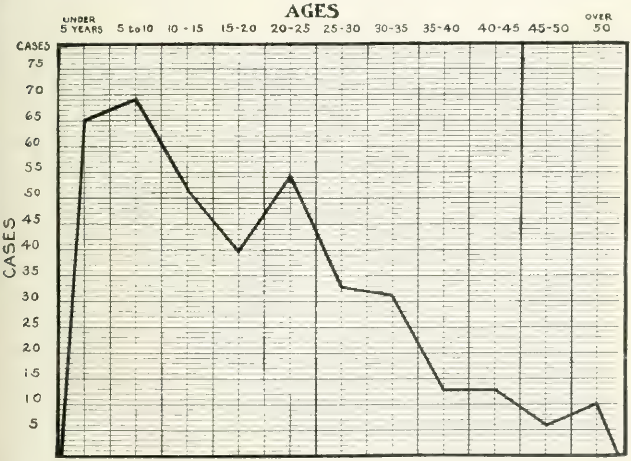
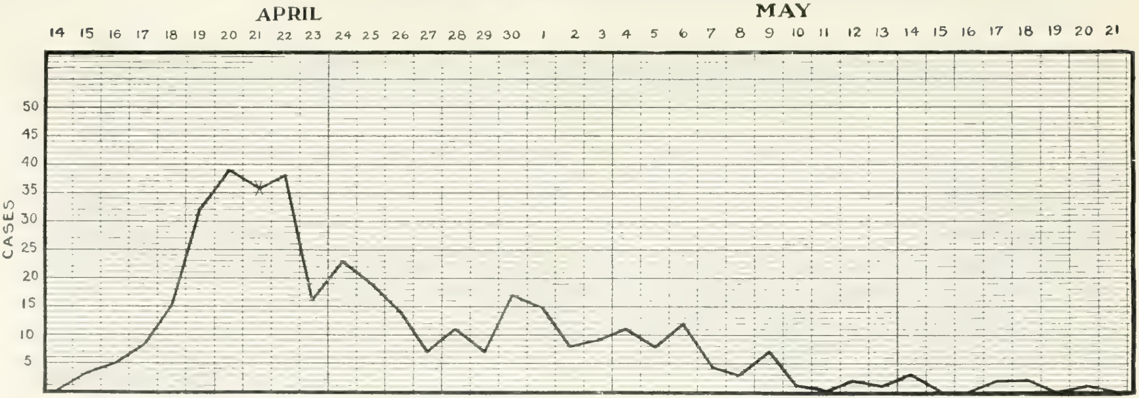


CHART 3.

SHOWING NUMBER OF CASES OF TYPHOID FEVER REPORTED EACH DAY DURING THE STAMFORD OUTBREAK.



SHOWS DATE ON WHICH THE IMPLICATED DAIRY WAS CLOSED. NOTE THAT AFTER FIFTEEN DAYS THE EPIDEMIC WAS PRACTICALLY AT AN END.

EXPLANATION OF DIAGRAM I.

The large square M N O P represents the town of Stamford.

B is the dairy distributing the implicated milk, and the dash lines running from B into the city represent the milk route of this dairy. Each of the dots represents one case of typhoid fever and is placed upon the route of the dairy from which it was supplied with milk. There are 368 such cases on B's route, including the 12 around the $\boxed{S}$, which is meant to represent the café supplied by B. B supplied about one-eleventh of the milk used in the town.

H H and H are distributing dairies similar to B.

C H and E B L are producing farms selling milk to B and also peddling some themselves. The dash line extending from E B L represents his personal route of 5 houses in which 8 cases of typhoid occurred.

J H B and J B H are producing farms selling milk to B and also to distributing dairies H and H H.

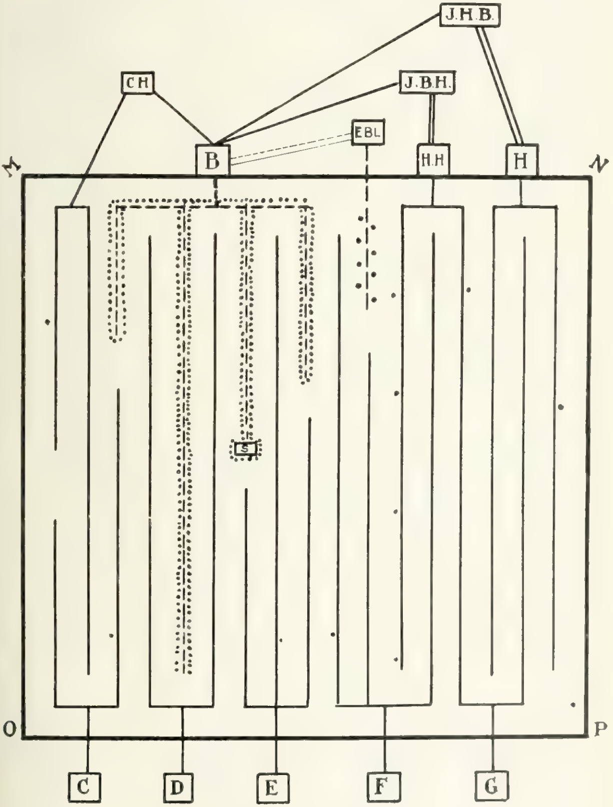
The double lines show the dairy to which the producer sold most of his milk.

Dash lines show the apparent course of the infective agent.

C, D, E, F, and G are other dairies having routes in Stamford.

DIAGRAM I.

SHOWING RELATION OF MILK ROUTES TO TYPHOID FEVER CASES DURING THE EPIDEMIC AT STAMFORD, CONN., 1895.



cases. B. obtained the milk sold by him from other parties and produced none himself. He was supplied regularly by 3 producers. E. B. L., C. H., J. B. H., and after April 12 also by J. H. B. C. H. besides furnishing milk to B. also supplied some in town himself, and among his customers only one case occurred. J. B. H. produced 4 cans of milk a day; one can went to B. ad 3 cans to dairyman H. H., on whose route occurred only 5 cases of typhoid. E. B. L. furnished B. from 140 to 150 quarts of milk daily; this constituted over one-half of B.'s supply. In fact, all that E. B. L. produced went to B., except a few quarts which he distributed to 5 families. It is significant that in these 5 households there were 8 cases of typhoid.

B.'s dairy was situated in a low, poorly drained part of the city. The water used to wash cans was from an uncemented dug well with a loose board cover 6 inches above the ground level. The well was 13½ feet deep and the water stood within 1 foot 9 inches of the top. There was a shallow, foul privy 25 feet west of the well on slightly higher ground, and another 40 feet to the east. The water supply was therefore a shallow surface well, uncemented, in poorly drained soil and in close proximity to two privies. Chemical and bacteriological examination of the water showed gross pollution. The last act in the washing of milk cans by B. was to rinse them in cold well water and invert them to drain and dry. The next morning these cans were taken to the producing farms for use. B.'s method of delivery was such that there was no part of his route which might not have received milk from the E. B. L. farm. B. washed all the cans coming to him and returned them clean to the producers. Farmer C. H. scalded the returned cans before refilling. E. B. L. refilled the cans just as they came from B., all of his milk going into them, including that which he delivered to his 5 personal customers. J. B. H. refilled cans returned from B. without any extra treatment. He had, however, in use 8 cans, one of which was returned daily from B., and 3 taken to H. H. No precautions were taken to keep separate the cans coming from the two dealers. J. H. B. did not begin to furnish milk to B. until after the outbreak was well started and H., who handled most of his milk, had only one case on his route.

No case of typhoid was found at the dairy or producing farms, but the hypothesis that the well water at dairy B. was infected would explain all the features of the epidemic, and whatever the source of the infection the fact remains that the disease followed the milk of this one dairy, B., and of that distributed to the 5 houses personally supplied by E. B. L.

B. supplied about 225 households in which 352 cases occurred, a café among the frequenters of which 12 cases developed, a bakery in whose patrons 2 cases were found, and 2 other fever patients were reported who had obtained this milk in other ways.

SCARLET FEVER.

No organism has as yet been isolated which is generally accepted as the specific cause of scarlet fever. In 1882 Mr. W. H. Power[a] investigated an outbreak diagnosed as scarlet fever which he believed was caused by infectious matter from a cow which had recently calved. In 1885 Power[b] investigated another epidemic which was practically limited to users of milk from a certain dairy at Hendon where several diseased cows with an eruption of the udders were supposed to have been the source of the infection. Klein[c] isolated from the lesions in the cows and also from human cases a micrococcus which he believed to be the specific organism of the disease and probably the cause of scarlet fever. This view has not been accepted. Sir George Brown,[d] who also investigated this outbreak, was of the opinion that the cow disease was possibly vaccinia, and that the milk had probably become infective by contact with a human case. Other similar outbreaks have subsequently occurred among cows without a corresponding epidemic among the users of the milk.

In the scarlet fever outbreaks which appear later, the abstracts were made from the reports cited, and the writer is aware that in a few of the cases the evidence is not entirely conclusive. In two of the cases the source of the infection is given as supposedly diseased cows. This is necessarily an opinion of the reporter and not a statement of fact, and these outbreaks have been included because the association of the disease to milk distribution was such as to make it probable that the milk, if not the carrier itself, stood at least in some relation to the carrier of infection, whatever the original source might have been.

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[a] Power (W. H.). Report of Local Govt. Board, Lond. (Medical Officer's Supplement), 1882, p. 65.

[b] Power (W. H.). Report of Local Govt. Board, Lond. (Medical Officer's Supplement), 1885, p. 73.

[c] Report of Local Govt. Board, Lond. (Medical Officer's Supplement) 1887–88, p. XIII.

[d] Report on Eruptive Diseases of the Teats and Udders of Cows in Relation to Scarlet Fever in Man, Agricultural Department, Privy Council Office London, 1888.

SUMMARY OF EPIDEMICS.

Of the 51 scarlet fever epidemics reported as spread by milk, compiled by the writer, 25 occurred in the United States and 26 in Great Britain; all cases enumerated in the outbreak were reported as living in houses supplied with the suspected milk in 27 of the epidemics; a case suffering from the disease at such a time as to have been the possible source of infection was found at the producing farm, the distributing dairy, or milk shop in 35 cases; the outbreak was supposed to have been due to bottles returned from infected households and refilled without previous sterilization in 3 cases; the diseased person or persons were mentioned as handling the milk or milk utensils in 3; the sick milked the cows in 12; the same person nursed the sick and handled the milk in 1; same person nursed sick and milked cows in 1; the outbreak was supposed to be due to disease of the cow in 2; it was reported that measures taken upon the presumption that milk was the cause of the epidemic were followed by abatement of the outbreak in 22 cases.

The following outbreak is one of many interesting illustrations:

SCARLET FEVER IN NORWALK, CONN.[a]

In November, 1897, an unusual number of cases of scarlet fever occurred in Norwalk. Population of Norwalk, South Norwalk and East Norwalk, 22,000. Previous to October 25 scarlet fever had been reported as follows: August, no cases; September, 5 cases; October 10, one case. The source of infection in most of these cases had been traced. Between October 25 and November 9, 29 cases developed. The 29 cases were distributed in 25 families and 24 houses. School infection was eliminated. Many cases did not attend school, and some were in families where they had no school children. The cases were widely separated; 17 of the infected houses were in South Norwalk, 3 in Norwalk, and 4 in East Norwalk. The families were of different social positions and contact-infection seemed improbable. The only factor in common to practically all of the cases was the milk supply. Twenty-seven out of the 29 obtained milk from one dealer, H. The other two were in one family in East Norwalk; they were a girl of 12 and boy of 9 years, and were taken ill on November 7 and 9, respectively. They had no connection with the milk route, nor could their infection be traced to any source.

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[a] Smith, (Herbert E.); Report Connecticut State Board of Health, 1897, p. 259.

The estimated daily supply of milk in Norwalk was 3,500 quarts. Dealer H. furnished 450 quarts, or about one-eighth of the whole, whereas he had twenty-seven twenty ninths of the scarlet fever cases on his route.

H. bought his milk from three producers. There were no cases of disease in the family of the milk dealer nor in those of two of the producers, A. and B. but on the third producing farm, K., a case of scarlet fever was found. This farm was in the Bald Hill district. The district school had opened September 7 with a registration of 23 pupils. On September 20 one of the pupils fell ill with scarlet fever; other cases followed, and the school was closed October 19. In all there were 20 cases, all in school children or in those coming in contact with them. Two of the above cases, living near farm K., were exceedingly mild and frequently visited and played at this farm with K.'s son, a lad of 4 years. This son broke out with a scarlatinous rash October 24.

Milk from this farm was carted to Norwalk and all of it sold to, and delivered by, dealer H., who placed the cans of milk from K. in his wagons with that from the other two producers, A. and B. No attempt was made to keep the cans separate, and, therefore, one day part of his customers might receive K.'s milk and the next day it would be delivered to others. H. supplied about 300 families, of which 24 were invaded. The sale of this milk was stopped November 7. The number of cases and the dates on which they occurred would indicate that the milk was not continuously infected. During the outbreak several cases of sore throat occurred among users of H.'s milk, which may possibly have had some casual relation to the infectious milk.

It would seem that cases of scarlet fever belonging to a school outbreak and visiting a dairy farm, and possibly also the boy on the farm, infected from his playmates, were the source or sources rendering the milk infective. The relation here of the two outbreaks is of interest, the one spread by school contact being the original source of the milk epidemic.

DIPHTHERIA.

Diphtheria epidemics apparently due to milk began to be reported in 1877 and 1878 in England. In certain cases the suspected milk came from herds where cows were found suffering from an eruptive disease of the udder, and this was thought to be the source of the infection. In this connection Klein[a] conducted some experiments on cows with the Klebs-Löffler bacillus. He took healthy milch cows and inoculated them subcutaneously in the shoulder with 1 cubic centimeter of a broth culture of the Klebs-Löffler bacillus taken from a

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[a] Klein, Report Med. Officer, Loc. Govt. Board, London, 1889, p. 167.

EXPLANATION OF DIAGRAM II.

A, B, and K are dairy farms selling their product to retail milk dealer H. K is the farm on which a case of scarlet fever occurred antedating the outbreak in Norwalk.

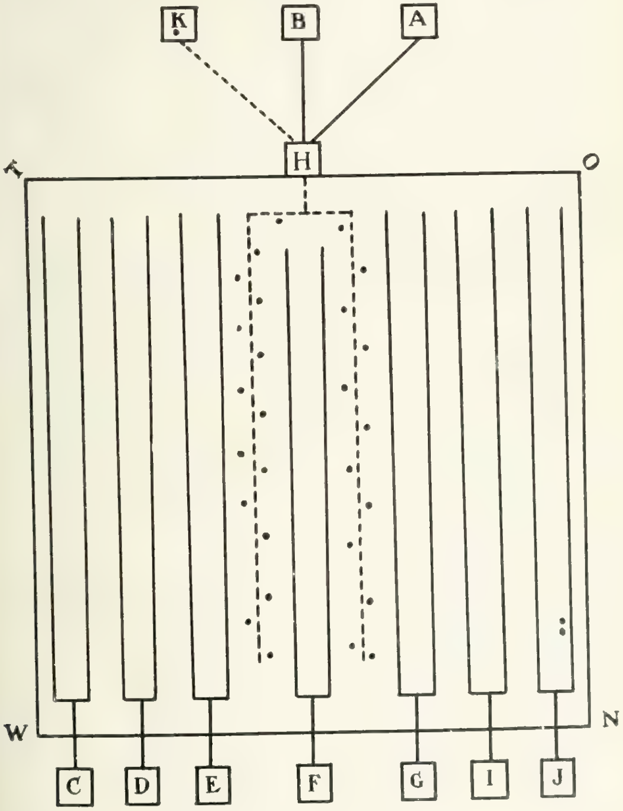
The large square T O W N represents the city of Norwalk.

H is the retail milk dealer among whose customers all cases but two occurred. The dash lines represent H's milk route, and each dot is a case of scarlet fever.

C, D, E, F, G, I, and J are other dairymen having routes in Norwalk. The lines extending from them into the city represent their milk routes and are introduced to show their freedom from the disease.

DIAGRAM II.

SHOWING RELATION OF MILK ROUTES TO SCARLET FEVER CASES DURING OUTBREAK AT NORWALK, CONN., 1897.



human case. These cows became ill, had a rise in temperature, and on the fifth day there appeared upon the udder an eruption characterized by papules, vesicles, and crusts. He states that he isolated the B. diphtheriae from the vesicles, pustules, and milk. Other experimenters[a][b] have however failed to get similar results. The Klebs-Löffler bacillus has been isolated from market milk by Bowhill,[c] Eyre,[d] Klein,[e] and Dean and Todd.[f]

KLEBS-LÖFFLER BACILLI IN MILK.

Dean and Todd reported that in certain families supplied with milk from two cows there occurred 2 cases of clinically typical diphtheria and 3 of sore throat, that in one family using the milk only after sterilization no case occurred. Inspection of the cows showed papules, crusts, and ulcers on the teats and udders. One of the cows seemed well and gave apparently normal milk; the other had a mammitis and gave a scanty, ropy, semipurulent and slightly blood-tinged milk. Cultures were made from the throat of one of the diphtheria patients and also from the ulcers and milk of each cow, and typical Klebs-Löffler bacilli were isolated in all cases. The milk of the cow with mammitis also contained streptococci. The bacillus isolated was virulent and markedly pathogenic to guinea pigs, but diphtheria antitoxin protected guinea pigs against large doses. The udder eruption was shown to be contagious to cows and capable of spread by the hands of the milker, but no B. diphtheriæ were found in vesicles and ulcers of the secondary bovine cases. Calves were not protected from this disease by diphtheria antitoxin, nor by this disease from cowpox. The conclusions drawn were that the ulcers on the udders had become secondarily infected with B. diphtheriæ, probably accidentally from some apparently healthy throat, and that the udder affection was a separate disease.

Eyre[g] has shown the ability of the B. diphtheriæ to proliferate in raw milk drawn from the cow under aseptic conditions as follows:

	0 hours.	24 hours.	48 hours	7 days.
B. diphtheriæ	39	1,170	22,000	19,000,000

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[a] Abbott (A. C.), Jour. Path. & Bact., 1894, II, p. 35.

[b] Ritter, Centralblatt f. Bakt., Referat, 1896, XIX, p. 662.

[c] Bowhill, Veterinary Record, 1899, April 8th.

[d] Eyre, Brit., Med., Jour., 1899., II, p. 586.

[e] Klein, Journal Hygiene, Camb., 1901, I, p. 85.

[f] Dean & Todd, Jour. Hygiene, Camb., 1902, II, p. 194.

[g] Eyre, loco citato.

SUMMARY OF EPIDEMICS.

Of the 23 diphtheria epidemics reported as spread by milk and compiled since 1895, 15 occurred in the United States and 8 in Great Britain; cases of the diseases occurred at the producing farm, distributing dairy or milk shop at such a time as to have been the possible cause of the outbreak in 18 cases; the diseased person milked the cows in 4; the same person nursed the sick and handled the milk in 1; the outbreak was supposed to be due to disease of the cows in 2; all cases of the disease were reported as living in households supplied with the suspected milk in 15 instances; measures taken upon the presumption that milk was the carrier of infection were reported as followed by subsidence of the outbreak in 5 cases; the Klebs-Löffler bacillus was isolated from the suspected milk in 2 of the epidemics.

The following outbreak is one of many interesting examples:

OUTBREAK OF DIPHTHERIA IN DORCHESTER, MILTON, AND HYDE PARK.[a]

On April 13, 1907, after a period of comparative freedom from diphtheria, there were reported to the board of health of the town of Milton 11 cases of that disease. This sudden explosion caused very naturally a feeling of grave apprehension on the part of the local health authorities. The following is an account of the epidemic: Cases of diphtheria were reported in Milton as follows: April 12, 1 case; 13, 11 cases; 14, 1 case; 15, 4 cases (of these 4 cases, 3 were in the same house and secondary to a case which had developed before the 12th and can therefore be considered as not belonging to this explosive outbreak); 16, 1 case. In Dorchester cases were reported as follows: April 12, 6 cases; 13, 19 cases; 14, 11 cases. In Hyde Park the number and dates were: April 13, 2 cases; 14, 5 cases; 15, 6 cases; 16, 1 case; 17, 3 cases, and 19, 1 case.

The following table shows the relationship of the cases in the different places:

Place.	April.									Total by towns.
	11.	12.	13.	14.	15.	16.	17.	18.	19.	
Milton		1	11	1	4	1				18
Dorchester		6	19	11						36
Hyde Park			2	5	6	1	3		1	18
Total		7	32	17	10	2	3		1	72

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[a] Monthly Bulletin, State board of health, Mass., May, 1907, vol. 2, No. 5, p. 117.

EXPLANATION OF DIAGRAM III.

J H, R B N, E T T, O H, J M B, and C F J are the farmers producing milk.

A is the milk dealer delivering milk in both Milton and Dorchester. B is the milk dealer delivering milk in Hyde Park.

The lines connecting the producing farms and the milk dealers show to which dairy the farmer sold his milk.

The large squares represent Milton, Dorchester, and Hyde Park.

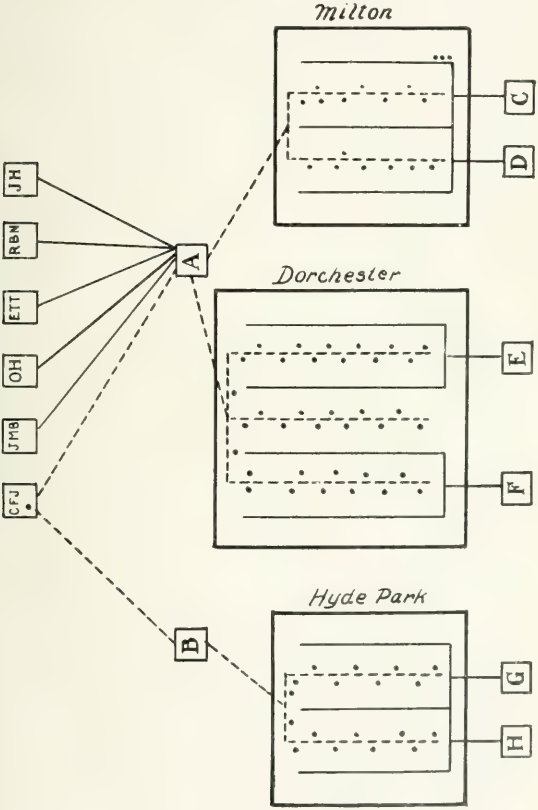
The dash-lines extending from A to B into the towns represent the milk routes carrying the supposedly infectious milk.

Each dot represents a case of diphtheria and is placed on the milk route from which it was supplied.

C, D, E, F, G, and H represent the other dairies selling milk. The lines extending from them into the towns represent their routes and are inserted to show their freedom from diphtheria cases.

DIAGRAM III.

SHOWING RELATION OF MILK ROUTES TO DIPHTHERIA CASES DURING THE OUTBREAK AT DORCHESTER, MILTON, AND HYDE PARK, 1907.



Investigation showed that all the cases in Dorchester and Milton were supplied with milk by one dairy, A, with the exception of 3 which were all in one house in Milton and were secondary to a case reported before the onset of the outbreak. In Hyde Park all the cases obtained milk from dairyman B. Dairy A bought its milk from 6 producers: J H, R B N, E T T, O H, J M B, and C F J (see diagram III). On none of the producing farms were any cases of diphtheria found except on that of C F J, where it was discovered that a child had been seized with the disease on April 11, and that the cooler in which the milk was mixed was washed in the house and that this office was performed by the person who had the care of the sick child. This same producer, C F J, also sold about one-third of his output to dairyman B, who delivered it in Hyde Park. Prompt action on the part of the local authorities in excluding sale of milk produced by C F J, brought the outbreak to an immediate close.

It will be noticed from the table that the outbreak in Hyde Park occurred a day later than that in Dorchester and Milton. This is explainable by the fact that B called for his share of C F J's milk in the evening and sold it on the following day, whereas A came for his in the morning and disposed of it at once.

It is of further interest that C F J himself came down with the disease after the outbreak had nearly subsided, and that dealer A's son who drank milk from C F J was one of the earliest victims.

EPIDEMICS OF SORE THROAT AND PSEUDO-DIPHTHERIA.

Among the collected epidemics are 7 variously reported as sore throat, pseudo-diphtheria and septic sore throat. They all occurred in Great Britain. Two of the outbreaks were supposed to be due to milk coming from cows having mastitis, 1 to milk of cows afflicted with teat and udder eruption; in one case the sequence was first severe sore throat, thought to be quinsy in the farmer, followed by mastitis in the cows and sore throats in other persons on the farm, and last an outbreak of sore throats on the milk route. Precautions taken against the milk were reported as stopping four of the outbreaks.

CHARACTER OF MILK EPIDEMICS.

Milk epidemics have characteristics more or less peculiar to themselves and usually show the following features:

(*a*) *Explosive onset.* The onset is usually sudden and may very aptly be termed explosive. This is due to the fact that a certain can or lot of milk receives an amount of the infective material from contact with an infectious person, premises, or water. This milk may be delivered to the consumers by itself, in which case the number of persons exposed to the infection is small. Or it may be mixed in a dairy with that from many other cans, and thus a larger amount of more dilute infectious material be delivered to the community. If kept cool, the milk may remain thus dilute as regards the pathogenic organism and the disease may crop out among the consumers only in those most susceptible or in persons drinking a comparatively large amount. But if the milk becomes warm because of lack of care or long transit and the contained organism is such that it will proliferate in milk, each quart delivered to the consumer may be more infectious than the original can. In either case the users of the infectious milk will receive their dosage of the organism at approximately the same time and will therefore, making due allowance for the normal variations in period of incubation, fall ill simultaneously.

The initial explosion may therefore consist of but a few cases if the amount of infected milk is small or if very dilute; or of many if the amount of infected milk is large or the number of organisms great. If the milk is infected at but one milking, the outbreak will rapidly subside and, aside from secondary cases spread by contact or other means, no new ones will appear. If the milk is infected day after day, the outbreak continues; and in contagious diseases, after the lapse of the period of incubation from the initial outbreak, secondary cases due to contact are apt to appear and grow more numerous, so that the picture presented of a typical milk epidemic may become less clear. A milk epidemic is therefore most typical in its onset, although under efficient systems of notification and quarantine secondary contact cases may be largely prevented and it may then maintain its characteristics throughout. The development of these secondary cases contracted by contact and otherwise explains why in the epidemics reported later in tabular form not all of the cases in the outbreak are reported as consumers of the suspected milk. Another explanation is that in most cities there are always a certain small number of cases of the commoner contagious diseases which have varied sources of origin and may be termed residual. It is on top of this as it were that an epidemic occurs.

(*b*) *Disease follows the milk.*—Disease carried by milk can occur only among users of the infectious milk. Milk routes may therefore at times be considered thoroughfares of infection. During an epidemic other cases of course may occurr among nonconsumers, but the contagion is carried to these by other means. In typhoid fever for example it is possible to conceive a water and a milk epidemic occurring at the same time, or what is possibly more common in the cities, during an unusual prevalence of typhoid due to various and in some cases unknown causes, smaller milk outbreaks may occur.[a]

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[a] Bull. No. 35, Hyg. Lab., U. S. Pub. Health and Mar.-Hosp. Serv., Wash., p. 20.

But milk epidemics necessarily follow the milkman, and often his route can be plotted by the incidence of the disease. The outbreak may be limited to a certain section of the city if the route is small and circumscribed in extent. This will usually eliminate water as a cause of typhoid where a public supply is in use. Or where various water supplies are used the cases may occur among the users of the different sources. If the dairy is a large one, delivering to all parts of the city, the cases may be widely separated and much scattered, having nothing in common but the milk supply. The children may go to different schools, the families be of varied social status. These points will usually eliminate schools and contact as sources.

At times where the area covered by the milk route and therefore the district involved in the outbreak is circumscribed, occasional isolated cases will be found at a distance, and upon careful investigation it will be found that they had friends or relatives on the involved route and used the suspected milk while on a visit.

Very interesting cases have been reported where the evidence seemed quite convincing of persons drinking a single glass of the suspected milk and falling ill after a due period allowed for incubation.

Milk outbreaks are as a rule more typical in small towns where the organization is less complicated and fewer extraneous factors occur to conceal the true picture. An example of this is the outbreak at Elkton, Md., in 1900.

Elkton epidemic.[a]—Elkton had a population of 2,542. The town water supply was obtained from the Elk River about 1½ miles above the town. Part of the families drank the town water, the rest used private wells. The inhabitants were supplied with milk from 4 dairy farms having routes in the town. Dairyman B on his way to town each day with his own milk obtained an additional amount from 2 other farmers, C and D, both of whose farms remained free from typhoid. In September, 1900, a case of typhoid fever occurred on farm A (see diagram IV) adjoining farm B. Mrs. B, wife of the dairyman, assisted in nursing the case at A for two or three weeks up to October 5. For some days before this Mrs. B and one of her sons had been ailing, but the boy continued milking and the mother handling the milk up to October 8, when both became too ill to work. (Later another son fell ill.) Previous to this time there had been in Elkton only 3 cases of typhoid and they were all in one family, occurring August 12, September 12 and September 19. On October 11, 3 cases of typhoid fever were reported; 12, 1 case; 13, 2 cases; 14, 3 cases; 15, 3 cases; 16, 3 cases; 18, 6 cases. By October 28, 32 families had been invaded. All used milk supplied by B, 18 used the town water supply, and 14 private wells. The total number of cases was 39. On this day B stopped selling milk and in three weeks the epidemic subsided. The final summary of the outbreak was: Invaded houses, 39; all used B's milk, 21 used public water supply, and 18 used private wells. B claimed to supply regularly 80 houses with milk. One hundred and eighty people lived in the 39 invaded households.

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[a] Fulton (John S.) Jour. Hyg., Camb., 1901, 1, p. 422.

There were several occurrences during this outbreak of special interest. Miss M, living in New Jersey, visited Elkton for two days, October 5 and 6, returning home on the 7th. While in Elkton she was at a house supplied with milk from B's farm. No typhoid had occurred at this house up to that time. On October 14 Miss M fell ill with typhoid. In one family a negro servant, whose chief food consisted of oatmeal and milk, left Elkton the middle of October and went to Glasgow, Del., where she became ill of typhoid and died. In another family was a married daughter who left Elkton the last of October to visit friends. In about ten days she fell ill with typhoid. At the jail where there were from 15 to 20 prisoners who received no milk whatever, 3 members of the jailer's family, and 2 men assisting about the place, all of whom used B's milk in one form or another, fell ill with typhoid, while the prisoners were not attacked.

In cities where large dairies are the rule, receiving milk from perhaps hundreds of farms some of which are situated miles away, it is necessarily very difficult at times to find the infecting focus. Cases of the disease may occur on two or three milk routes, and search will show that they all receive part of their milk from the same farm or else that one dairyman at times sells surplus milk to the others, but the milk consumed will all be directly or indirectly traced to some common source of contact of disease with the milk. In tracing the relationship between milk and the disease, ice cream and other forms of milk preparations such as whipped cream are to be borne in mind. A confectioner's shop or bakery may be the focus producing an epidemic.

(*c*) *Special incidence in milk drinkers.*—In addition to the fact that as a rule cases occur only in houses using the infectious milk, many times interesting incidents occur where in a family the only person attacked will be one drinking raw milk, or where the only person exempt will be the sole one not using it. Usually cases are found mainly among the milk drinkers.

(*d*) *The better houses suffer greater invasion.*—The so-called better class of houses are often attacked in greater proportion than others. This is explained by the fact that families with larger incomes are supposed to drink more milk, whereas those with lesser resources use it mainly in tea or coffee or cooked in food preparations and for children.

EXPLANATION OF DIAGRAM IV.

Each red dot represents a case of typhoid fever.

A—Farm where original case occurred in September and was nursed by wife of farmer B.

B—Dairy farm where wife nursed preceding case and prepared the milk for market. She and one son were ailing for some days but did not stop work until October 8.

The dash-lines represent the course and distribution of the milk from farm B. All the cases of typhoid were on this milk route.

C and D were farms selling milk to farmer B. No typhoid occurred on these 2 farms.

E—Farm receiving a small amount of milk daily from B for use of girl staying at farm. This girl contracted typhoid.

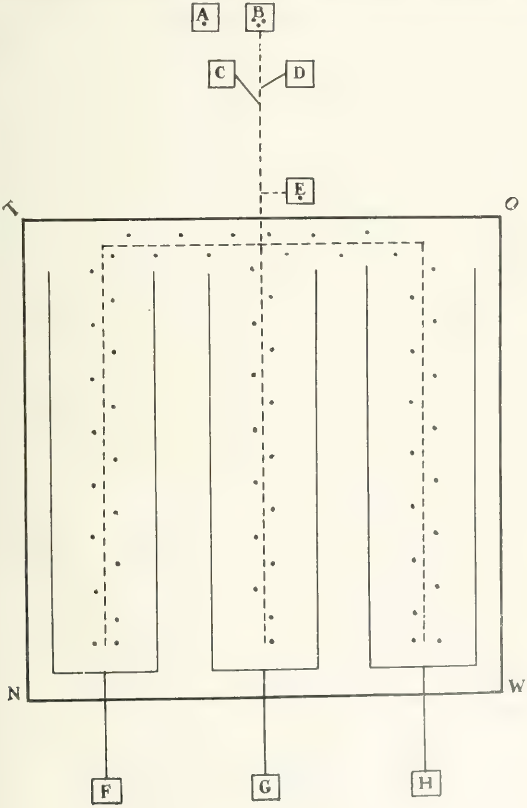
F, G, and H—The 3 other dairy farms supplying milk to Elkton. The solid lines represent their routes. No case of typhoid on these routes.

Population of Elkton	2,542
Total cases	64
Houses invaded	39
Invaded houses using B's milk	39
Invaded houses using well water	18
Invaded houses using town water	21

The large square—"TOWN"—represents the town of Elkton.

DIAGRAM IV.

SHOWING RELATION OF MILK ROUTES TO TYPHOID FEVER CASES AT ELKTON, MD., 1900



Among the well to do therefore it frequently happens that infectious milk finds more victims, while among the poor the children are the ones most likely to suffer.

(*e*) *Age and sex.*—Women and children are usually credited with drinking more milk than men, and it is generally believed that a greater incidence of the disease in them is a characteristic of milk-borne outbreaks.

BACILLUS CARRIERS.

The term "bacillus carrier" is most commonly associated with carriers of typhoid or Klebs-Löffler bacilli, and is used to designate persons who discharge the former in their feces or urine, or both, or harbor Klebs-Löffler bacilli in their nose or throat. They may be acute or chronic carriers depending on whether they carry the organisms for short or long periods of time.

Diphtheria carriers may become such from having had an acute attack of the disease, or by associating with others having acute attacks, or with other bacillus carriers. Klebs-Löffler bacillus carriers have undoubtedly frequently infected milk, and thus produced epidemics of milk diphtheria. This in all probability is more likely to happen when the carrier is a milker at a dairy farm.

Typhoid carriers are of particular interest because it has been found that an appreciable number of typhoid convalescents[a] discharge typhoid bacilli in the urine or feces, or both, and that from 2 to 4 per cent continue to do so and become chronic typhoid bacillus carriers, that some continue so for years and some during the remainder of their lives. It is now known that not only may convalescents become carriers, but that nurses and those coming in contact with the sick or with other carriers may in turn become typhoid bacillus carriers for longer or shorter periods of time and that, at times, without themselves falling victims to the disease.

Undoubtedly these bacillus carriers constitute one of the important factors in the spread of typhoid fever by milk. Individuals in the early stages of typhoid may be physically well enough to continue at work milking or handling milk; others with very mild attacks may not cease work at all. Both may be discharging typhoid bacilli in the excretions. On the other hand, the chronic typhoid bacillus carriers may continue to discharge bacilli not only for weeks but for years, and being well, remain at work and continue a menace over long periods of time.

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[a] W. T. Graham, C. L. Overlander, John E. Overlander, and M. A. Dailey found 23 per cent. (Boston Med. and Surg. Journ., Jan. 14, 1909.) Officers of the medical and sanitary departments of the Government of India at the Central Research Institute at Kasauli found 11.6 per cent. (Scientific Memoirs, No. 32, "An enquiry on enteric fever in India," p. 7.)

When it is considered that available evidence seems to show that between 2 and 4 per cent of typhoid convalescents become chronic bacillus carriers, the probability that some of them are employed at dairies in milking cows and handling milk is very great.

Several epidemics, due to milk infected by these chronic typhoid bacillus carriers, have been reported. Others will undoubtedly be found with increasing frequency as epidemics are studied with this possibility in mind.

Dr. Henry Albert reports a small but interesting outbreak of this kind occurring in the autumn of 1907 at Cedar Falls, Iowa:

> A certain gentleman had typhoid fever a year ago and recently 4 cases of typhoid fever developed in his own family, 7 in the family of one neighbor and 2 in the family of another neighbor. The man who had typhoid fever a year ago owned a cow, did his own milking, and supplied milk to the 2 families in which the cases, respectively, 7 and 2, developed. The man who is supposed to be the source of this infection is apparently perfectly well, but has a slight cystitis and on the examination of his urine, typhoid bacilli were isolated. The water used by this man and his family came from a rather shallow well. It contained a large number of Colon bacilli, but no typhoid bacilli were found. This water was, however, not used by any member of the other 2 families. Just how the bacteria gained entrance to the milk, whether from the hands of the bacillus carrier or from the water used for cleaning milk pails, is difficult to determine, but it seems very certain that the milk was the medium through which the infection of the 9 cases in the neighboring families was carried.

Branthwaite reports an outbreak at Brentry Reformatory, an institution for the detention and treatment of habitual inebriates. The reformatory consisted of 16 buildings, scattered over about 98 acres of ground. The institution was practically isolated from other communities and housed usually about 265 persons. It ran its own dairy. From 1899 to 1906 the institution remained free from infectious diseases. The first case of typhoid occurred in September, 1906, and by November, 1907, 28 cases had developed. The patients fell ill at irregular intervals and always in groups of from three to five. The first patient was a woman who had been in the institution several months, and therefore removed from the possibility of outside infection. Careful search was made as to the possible introduction of the disease, but in spite of all precautions others continued to be attacked. A detailed investigation pointed to the milk as the carrier of infection. All milk had been regularly sterilized, and therefore if it were the cause it was necessarily due to defective methods or to subsequent contamination. A new sterilizing plant was installed, and all handling of milk after heating was limited to two persons, a man and a woman. But still cases developed, and all evidence continued to point to the milk. It was therefore concluded that infectious material reached the milk after sterilization, and it was decided to find whether one of the two persons handling it was a bacillus carrier. The woman milk handler was removed from the dairy and her feces examined. Pure cultures of the typhoid bacillus were isolated from her stools. She was permanently removed from the dairy, and, after a lapse of the usual incubation period of two weeks, no new cases had developed up to the time of the report, three months later.

Dr. A. K. Chalmers, medical officer of health of Glasgow, Scotland, reported an outbreak in Glasgow in December, 1907, and January, 1908. This outbreak is of interest because of the parts played by a bacillus carrier and an acute case of typhoid fever in the production of the epidemic. There were 126 cases of typhoid, in all of which the patients obtained milk from one dairy. Eight of the patients sickened between December 5 and 14. One of these 8 cases developed at Parkhouse dairy farm, which supplied milk to the distributing dairy from which all the other patients obtained their milk. Then for four days there were no cases. Following this, between December 19 and January 13, 93 cases developed. The result of much careful work in studying the relationship of the milk distribution to the incidence of the disease showed that the Parkhouse farm was the undoubted source of infection, and while the acute case which developed there could, from a standpoint of time and opportunity, have been the direct cause of the 93 and more cases which formed a typical milk epidemic, and undoubtedly was the cause, yet there remained to be found the source of infection of the first 8 cases, of which this patient was one. The water supply of the farm could not be found at fault, and so an investigation was made of the dairy hands. There was found an elderly woman milker who gave a history of having been previously associated with outbreaks of typhoid fever. She had had the disease sixteen years before. Her stools were examined and the typhoid bacillus isolated. Her blood gave a positive Widal reaction with "the laboratory strain of the typhoid bacillus." The conclusions drawn were that this woman was the source of infection of the first 8 cases occurring between December 5 and 14, one of which developed on this same farm in a woman who was ill from December 7 to 24, when her case was diagnosed as typhoid fever, and that this latter case was the source of infection causing the typical milk outbreak between December 19 and January 13.

Kayser, in 1905, reported two small milk outbreaks of typhoid fever traced to chronic bacillus carriers as the source of infection.[a]

SOURCE OF MILK CONTAMINATION.

(1) *From hands of milker.*—Many dairy employees take no precautions to keep the hands clean, and in fact the milker who washes his hands before milking is the exception and not the rule. He may be a typhoid bacillus carrier and be discharging typhoid bacilli in the excretions, and any carelessness in toilet is apt to deposit bacilli on the hands and under the finger nails. In the act of milking it is more than likely that he will wash at least some of them into the milk pail, and especially so if he resorts to a custom, all too common, of moistening his hands by squirting milk upon the palms preliminary to milking.

The milker's hands may have become soiled in acting as nurse for some case of typhoid in the family. He may be a convalescent from scarlet fever and be shedding particles of epidermis into the milk, or he may have diphtheria, or possibly tuberculosis, and with every act of sneezing and coughing spray tubercle or Klebs-Löffler bacilli with particles of sputum. If he does, as is not entirely unknown among careless milkers, and moistens his hands by spitting into the palms to facilitate the action of the fingers upon the teats, it is easily seen how infective material may find its way into the milk.

(2) *Air and dust of the stable.*—The stable dust may contain organisms eliminated by those working in it, and as some of this dust and other stable refuse adhering to the flanks, buttocks, and udders of the cows and floating in the air finds its way into the milk, under the conditions sometimes employed, it may carry with it these organisms.

(3) *The milk pail.*—The milk pail may have been washed and taken care of by some person or member of the family suffering from a contagious or infectious disease and in the handling have received its quota of typhoid or other bacilli which thus find their way into the milk.

(4) *Water supply.*—The water supply of the farm or dairy may be at fault. Farms are often very unfortunate in the location of their wells, which very frequently become polluted by cases of typhoid on the premises. The privy vaults are at times not far distant and are apt to be leaky and subject to seepage, and when a case of typhoid

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[a] A further discussion of the subject of bacillus carriers and epidemics due to them will be found in the chapter on "The milk supply of cities in relation to the epidemiology of typhoid fever."

fever occurs on the place or a person eliminating the bacilli sojourns temporarily on the premises, the possibility of water contamination exists. In some cases the dejecta of typhoid patients are buried in shallow holes around the house and often unwittingly around the well, while at other times, as occurred in some of the epidemics reported later, the dejecta were simply thrown on the ground where they could easily find their way into the water supply. Pails washed in polluted water, if not afterwards scalded, may contain the infective material and the more so if some of the last rinsing water still remains in them. The possibility of this water being added directly to the milk need not be considered, although it has undoubtedly played an important part in some epidemics. The water used may be a stream into which some household higher up empties its sewage. It has been supposed that cows wading into polluted streams might get upon the udders contaminated water, which in the act of milking would find its way into the pail. This at least is one of the rarer means of infecting milk.

(5) *Milk cooler.*—If a milk cooler is used and not properly taken care of, infectious material may reach the milk through it.

(6) *Cans.* If the milk is then put into cans the same possibilities are again met as in the pails.

(7) *Transportation.*—If the milk is now shipped to a distributing dairy in the city there is always the possibility of its infection in transit by those handling it, and it must always be borne in mind that some person may surreptitiously dip into the container with a soiled vessel or dipper or even drink from the mouth or top of the can.

(8) *Distributing dairy.*—Then there are the receptacles used by the retailer. In many distributing dairies the milk comes in by train in large cans, and before the contents are poured together in the mixer each can of milk must be tasted to ascertain whether or not it is sour. One man usually does the tasting. It may be done in a manner free from criticism or the taster may tip each can before it is lifted from the wagon and, removing the top, place his mouth to the can and taste the milk. When milk has been treated in this manner it has at times been the custom to draw into the mouth a sufficient amount and then spit it upon the ground. One taster has been mentioned who was so economical that he returned the tasted milk to the can. Another means of tasting which has at times been employed is to use a spoon or small dipper, inserting it into one can after another, and of course between cans into the mouth of the taster. A method less subject to criticism is to tip each can, then removing the cap, taste of the milk adhering to it. The cap can then be cast aside and scalded before

further use and the milk emptied into the mixing tank. Other methods entirely free from criticism are commonly used by careful dairies.

(9) *Bottles.*—It is at present the custom to deliver milk to the consumer in bottles. This is especially so in the cities. It can be seen how this practice properly operated may be better than any other; but, on the other hand, if carelessly conducted may be a source of much danger. Clean milk in sterile, well-capped bottles, handled and delivered by clean men, free from disease, is a condition much to be desired. But where empty bottles returned from the consumers' houses are not properly scalded before being again filled, the possibility of contamination by pathogenic organisms is necessarily considerable. Bottles left at houses where there are cases of scarlet fever, typhoid, or diphtheria, if refilled without being properly scalded, are undoubtedly a source of much danger. Many cities have ordinances to prevent this, but the constant presence of mild cases of disease, so mild and, according to present standards, atypical, that a correct diagnosis is not made, renders all regulating measures more or less ineffective. The accidental infection of bottles in an orderly, well-regulated household need not be considered so long as certain classes of people persist in using them for various other purposes, such as urinals and receptacles for sputum. Dr. Herbert Fox, chief of the laboratories of the Pennsylvania state department of health, states:

> The attention of the commissioner of health, Dr. Samuel G. Dixon, was called to a slimy mass of material on the under surface of a milk-bottle cap. He sent this to the laboratory and it was received in a very dry condition. Upon softening down and smears made from it we were able to obtain sufficient proof that it was sputum. Doctor Dixon informs me that he has known of milk bottles used for cuspidors on more than one occasion.

The practice of drinking directly from the bottles is a habit that must also be borne in mind as a possible means of contamination with tubercle and Klebs-Löffler bacilli. An example of apparent bottle infection is found in the typhoid outbreak at Montclair, N. J., in 1902.

Montclair epidemic.—During the summer and autumn of 1902 there was only an occasional case of typhoid in Montclair.[a] The 1st of December several cases occurred, apparently having milk from one dairy as the only factor in common. Investigation of the farms producing this milk failed to reveal any cases of disease which could be the source of the infection. All persons coming in contact with the milk were apparently in good health. More careful examination of the invaded houses showed that cases of typhoid existed only in those houses receiving milk in pint bottles. There were no cases among the quart-bottle customers. Cases continued to be reported on this route and the sale of milk from the dairy was therefore stopped. After two weeks new cases ceased to develop. It was then found that a man from New York City had come to Montclair ill with typhoid fever and had remained for a few days at a house supplied with milk from this dairy until he could be removed to a hospital. This house had during the patient's stay been supplied daily with three pint bottles of milk. The empty bottles were removed daily and, without sterilization, refilled and delivered to other houses. It seemed that this was the means of spreading the disease. Eighteen cases occurred in Montclair and 10 in Bloomfield, all in houses supplied with milk in pint bottles from this dairy.

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[a] Ninth Annual Report, Board of Health, Town of Montclair, N. J., 1903.

Pathogenic organisms may possibly reach the milk through dust while in the care of the vendor, but most likely the vendor himself is the more important and that, while organisms floating in the air can undoubtedly settle into milk, yet the chief danger is from contact with diseased persons or those having an intimate relation with the sick.

DETECTION OF MILK EPIDEMICS.

When in a city an unusual number of cases of scarlet fever, diphtheria, or typhoid fever occurs among the customers of any one dairy, it may be considered a sufficient reason for causing a careful inquiry to be made and a search for some source of milk infection. The mere finding of cases on one milk route is not by any means conclusive that milk is the carrier of the infection, but it is sufficient to cast suspicion and at times, undoubtedly, also to warrant regulation, even if no source of contamination is found, for it is often exceedingly difficult to find the infective focus.

The health officers of many cities have for some time been charging each case of typhoid fevor, scarlet fever, and diphtheria to the dairyman supplying the milk to the invaded household. In this way it is apparent when an unusual number occurs on one route, and measures can be taken to ascertain whether the incidence of the disease has an etiologic relationship to the milk. Cases which otherwise would show no relationship to each other are revealed as associated, and the milkman makes neighbors of families separated by considerable distance. In the complicated life of cities this gives the health officer a valuable aid in the control of certain of the common infectious diseases.

PREVENTION OF MILK EPIDEMICS.

Inspection and regulation of the production, handling, and sale of milk will lessen the number of milk epidemics. In cities the proper charging of each case of scarlet fever, diphtheria, and typhoid fever to the dairy on whose route it occurs will often reveal milk outbreaks, which can then be suppressed before reaching too great proportions. The most rigid inspection and regulation practicable at the present time, however, are impotent to prevent chronic bacillus carriers from being employed on milk farms and at dairies. They are also unable to keep mild ambulant cases of infectious diseases from being so engaged, for the reason that such cases can often not be diagnosed until after other cases have developed. Soper's case[a] of "Typhoid Mary" was a constant danger in her capacity as family cook to the members of the family in which she happened to be employed and to visitors eating of the salads and food prepared by her, but what might have happened had she been employed in the handling of milk distributed over a large city route can only be surmised.

The only way to prevent these epidemics entirely would appear to be to pasteurize or sterilize the milk, either at the dairy before delivery to the consumer or in the household after delivery.

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[a] Soper (George A.), Jour. Am. Med. Assn., June 15, 1907, p. 2019.

POINTS OF INTEREST IN REPORTING MILK EPIDEMICS.

In reporting milk epidemics some of the points of special interest are the following:

1. The number of cases of the disease existing in the involved territory during the time covered by the epidemic.
2. The number of houses invaded by the disease.
3. The number of invaded houses supplied in whole or in part, directly or indirectly, by the suspected milk.
4. The number of cases occurring in invaded houses so supplied.
5. The number of houses supplied with the suspected milk.
6. The relative proportion of houses so supplied to those supplied by other dairies.
7. The time covered by the epidemic.
8. The location of the case or cases from which the milk became contaminated.
9. The relation of the original case to the milk.
10. The time relation of the original case to the epidemic.
11. The special incidence of the disease among milk drinkers.
12. The elimination of other common carriers of infection.
13. The effect upon the epidemic of closing the dairy or taking such measures as will eliminate possibility of milk contamination from the suspected focus.
14. The finding of the specific organism in the milk.

BUSEY AND KOBER'S SUMMARY OF EPIDEMICS.

Busey and Kober summarized the epidemics compiled by them as follows:

TYPHOID-FEVER EPIDEMICS.

Mr. E. Hart tabulated 50 epidemics of typhoid fever and we have collected 88, making a total of 138 epidemics traceable to a specific pollution of the milk, the main facts of which are presented in a subjoined table. In 109 instances there is evidence of the disease having prevailed at the farm or dairy. In 54 epidemics the poison reached the milk by soakage of the germs into the well water with which the utensils were washed and in 13 of these instances (Nos. 5, 24, 39, 45, 70, 89, 90, 98, 99, 103, 111, 116, 124), the intentional dilution with polluted water is admitted. In 6 instances (Nos. 10, 74, 104, 107, 112, 121) the infection is attributed to the cows drinking or wading in sewage polluted water. In three instances (Nos. 118, 123, 131) the infection was spread in ice cream prepared in infected premises. In 21 instances the dairy employees also acted as nurses (Nos. 1, 6, 12, 16, 17, 24, 30, 37, 38, 41, 46, 52, 65, 68, 82, 110, 111, 115, 126, 127, 133). In 6 instances (Nos. 101, 102, 113, 117, 132, 134) the patients while suffering from a mild attack of enteric fever, or during the first week or ten days of their illness continued at work, and those of us who are familiar with the personal habits of the average dairy boy will have no difficulty in surmising the manner of direct digital infection. In one instance (No. 24) the milk tins were washed with the same dishcloth used among the fever patients. In one instance (No. 87) the disease was attributed to an abscess of the udder, in another (No. 92) to a teat eruption, and in No. 81 to a febrile disorder in the cows. Nos. 85, 103, 120, and 127 were creamery cases. In No. 96 the milk had been kept in the sick room.

SCARLET-FEVER EPIDEMICS.

Mr. Hart collected 15 epidemics of milk scarlatina, and we have tabulated 59, making a total of 74 epidemics spread through the medium of the milk supply, the details of which will be found in Table No. II.

In 41 instances the disease prevailed either at the milk farm or dairy. In 6 instances persons connected with the dairy either lodged in or had visited infected houses. (See Nos. 8, 9, 10, 11, 15, 40.) In No. 12 the milkman had taken his can into an infected house. In 20 instances the infection was attributed to disease among the milch cows; in 4 of these (Nos. 17, 18, 19, 35) the puerperal condition of the animal is blamed. In 9 instances disease of the udder or teats was found. (See Nos. 30, 31, 34, 39, 41, 59, 61, 62, 66.) In one instance (No. 54) the veterinarian diagnosed a case of bovine tuberculosis. In 6 instances there was loss of hair and casting of the skin in the animal. (See Nos. 17, 18, 19, 38, 40, 41.) In No. 68 the cattle were found to be suffering more or less from febrile disturbance. In 10 instances the infection was doubtless conveyed by persons connected with the milk business, while suffering or recovering from an attack of the disease (see Nos. 2, 22, 26, 29, 42, 57, 58, 60, 69, 71), and in at least 8 cases by persons who also acted as nurses. (Nos. 1, 2, 7, 9, 13, 14, 25, 63.) In three instances (Nos. 1, 73, 74) the milk had been kept in the cottage close to the sick room. In No. 15 the cows were milked into an open tin can which was carried across an open yard past an infected house, and in No. 53 the milkman had wiped his cans with white flannel cloths (presumably infected) which had been left in his barn by a peddler. Nos. 21 and 44 appear to have been instances of mixed infection of scarlet fever and diphtheria.

DIPHTHERIA EPIDEMICS.

Mr. Hart collected 7 epidemics of milk diphtheria and we have added 21 more. (See Table III.) In 10 of these 28 instances diphtheria existed at the farm or dairy, and in 10 instances the disease is attributed directly to the cows having garget, chapped and ulcerative affections of the teats and udder, while in No. 13 the cows were apparently healthy but the calves had diarrhea. (See Nos. 2, 5, 14, 18, 19, 20, 21, 22, 24, 25.) In No. 23 one of the dairymaids suffered from a sore throat of an erysipelatous character, and in No. 27 the patient continued to milk while suffering from diphtheria. In No. 28 one of the drivers of the dairy wagons was suffering from a sore throat.

MILK EPIDEMICS.

[Compiled by John W. Trask.]

TABLE I.- *Typhoid.*

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
1	October, 1907...	Cresson, Pa.......	9	0	9	Explosive outbreak in 8 families, no 2 of which used the same water supply. All took milk from the same dairy, at which 2 cases of typhoid were found in family of dairyman. Dairy closed and outbreak ceased.	At dairy.......	Rinsing cans with unboiled polluted well water prior to filling with milk.	Explosive outbreak limited to patrons of 1 dairy at which cases of typhoid were found. Outbreak ceased when dairy was closed.	Dr. J. A. Lynch, Cresson, Pa. Special report.
2	Oct.-Mar., 1907.	Cedar Falls, Iowa.	13	..	13	Mr. A had typhoid fever the year before. He owned a cow, did his own milking and sold milk to 2 neighbors. Four cases of typhoid developed in his own family, 7 in one of the neighbor's, and 2 in the other family supplied by him with milk. A was apparently well with the exception of slight cystitis. Examination of his urine showed the presence of the typhoid bacillus. The water used by A came from a shallow well and contained many Colon bacilli but no typhoid bacilli were found in it. This water was not used by any member of the neighboring families.	Chronic typhoid bacillus carrier who owned the cow and did the milking.	Possibly from hands of milker.	....	Dr. Henry Albert, professor of pathology and bacteriology, College of Medicine, State University of Iowa, Iowa City, Iowa. Special report.

MILK EPIDEMICS—Continued.

TABLE I. *Typhoid*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
3	September, 1907.	Somerville, Mass.	46	....	34	Explosive outbreak. Thirty-four cases supplied by 1 milkman, A, the others by 7 different milk dealers. A had during early part of September supplied milk in bottles to 2 houses having typhoid. Empty bottles were not sterilized.	Probably in house on milk route.	Probably by infected bottles.	Explosive outbreak limited largely to 1 milk route.	Dr. F. L. Morse, Somerville, Mass. Special report.
4	September, 1907.	Portland, Oreg.	54	......	5	Cases occurred on 1 milk route. Dairyman's daughter had typhoid.	Dairyman's daughter had typhoid.	....	....	Dr. A. L. Vial, Portland, Oreg. Special report.
5	September, 1907.	Lynn, Mass.	19	2	19	Explosive outbreak among consumers of milk from 1 dairy. Milkman's son had typhoid.	Milkman's son.	....	Explosive outbreak among consumers of milk from 1 dairy.	Dr. F. E. Stone, Lynn, Mass. Special report.
6	August, 1907.	Ansonia, Conn.	5	[illegible]	51	On Aug. 12 2 cases were reported. Within 48 hours 18 cases were reported, and investigation showed that all but 2 of these obtained milk from the same dairy. Analysis of water supply of dairy showed pollution with Colon bacilli.	On dairy farm (?).	Use of polluted water (?).	Epidemic ceased after proper action was taken against milk supply.	Dr. L. H. Wilmot, health officer, Ansonia, Conn. Special report.
7	July, 1907.	Coshocton, Ohio.	39	2	33	Explosive outbreak between July 13 and Aug. 4 among patrons of 1 milk dealer, at a time when other means of spread seemed absent. Dairy hand had typhoid. Dairy well polluted. Only 4 of the cases had never used the sus-	Case at dairy.	Might have been either in handling bottles or by using polluted well water.	Explosive outbreak limited largely to users of 1 milk supply.	Paul Hansen, assistant engineer Ohio State board of health. Special report.

						pected milk and these had been recently out of town. 3 had eaten it in ice cream. 22 of the cases developed within 6 days. Cases limited to territory covered by this 1 milk route.				
8	June, 1907	Wayne, Pa	42 (?)	....	42 (?)	Sudden outbreak among patrons of one dairy at which the proprietor had an undiagnosed case of typhoid. Dairy water supply was badly polluted; the milk contained many Colon bacilli.	At dairy	Probably by polluted spring water.	Sudden outbreak limited to patrons of 1 dairy.	Dr. H. H. Whitcomb, Norristown, Pa. Special report.
9	May, 1907	Savannah, Ga	95	....	63	Only 19 cases of typhoid fever had occurred in Savannah from Jan. 1 to May 1, 1907, and of these 2 were imported. During May 95 cases were reported, 63 of these took milk from one dairy A, the other 32 cases were scattered over 18 other milk supplies. Dairy A did a wholesale and retail business. One of the depots supplied by this dairy had a case of typhoid from Feb. 7 to Mar. 5. The milk depot was located in a baker's shop and the case of typhoid lived over the shop. Cans from this depot were returned unsterilized to the dairy and refilled with milk for other customers.	Lived over milk depot.	Possibly by infected cans returned from milk depot to main dairy.	The predominance of cases among patrons of 1 dairy.	John F. Anderson, passed assistant surgeon, P. H. and M. H. S. Special report.
10	January, 1907	Yardley, Pa	24	2	24	Yardley has population of about 1,000. In past 4 years has had 4 cases typhoid. There was an explosive outbreak of typhoid during the latter part of January and 1st of February, 1907, in which 22 cases were reported within a few days (about 3 weeks). All cases took milk from one man and no cases existed excepting among customers of this man. Ten of the 24 cases were under 15 years of age.	Probably on dairy farm. There had been a case there 2 years before. Water supply was badly polluted, shallow well receiving seepage from privy.	Most likely by polluted water used in washing cans and utensils.	Sudden explosive outbreak limited entirely to 1 milk route.	Dr. H. L. Bassett, president local board of health, Yardley, Pa. Special report.
11	1907.	Chattanooga, Tenn.	7	....	7	Outbreak on 1 milk route. A case of typhoid was found at the dairy. Dairy was closed and outbreak ceased.	At dairy	....	Outbreak limited to one milk route.	Dr. J. T. Shepherd, Chattanooga, Tenn. Special report.

MILK EPIDEMICS—Continued.

TABLE I. *Typhoid*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
12	October, 1906...	Newark, N. J...	103	..	69	Average monthly cases for Newark, 30 or less. Cases reported between Oct. 15 and Nov. 23 were 103, of which 69 were supplied with milk by a dairy receiving milk from a farm on which there had been a case of typhoid during latter part of September and first half of October. This case milked cows up to Oct 4. Well water was found to contain colon bacilli. Sale of this milk was stopped and during December only 49 cases were reported against 102 in November	On milk farm...	...	Explosive increase of cases 67 per cent of which had a common milk supply. Cessations of outbreak 2 weeks after sale of milk was stopped.	David D. Chandler, health officer, Newark, N. J. Reported by letter Oct. 9, 1907.
13	October, 1906...	Washington, D. C.	30	......	30	All circumstances pointed to milk as the carrier of infection. 2 cases of typhoid were found on farm producing milk	On dairy farm.	Milk was handled by persons coming in contact with sick.	Outbreak ceased when proper precautions were taken against milk	Dr. W. C. Woodward, health officer D. C. Special report
14	October, 1906...	Rochester, N. Y..	19	4	19	These 19 cases were reported between Oct. 5 and Oct. 31. Typhoid had previously been rare in Rochester. All cases were on the milk route of 1 milkman whose driver had a brother sick with typhoid and to whom he left milk in bottles daily. These bottles were never sterilized. With proper precautions taken against the milk the outbreak stopped.	On milk route in person of 1 of the customers of milkman.	Probably by infected bottles refilled with milk without sterilization and delivered to other customers.	Typhoid rare in Rochester. All cases on 1 milk route. Epidemic stopped when possibility of milk infection was removed.	Dr. J. Roby, Rochester, N. Y. Special report.

						Cases were widely scattered and other means of infection were eliminated.				
15	September, 1906.	Lynn, Mass	31	3	31	Explosive outbreak limited to consumers of milk from 1 dairy. Bottle washer at dairy had typhoid fever.	Boy at dairy. .	In washing bottles by boy in early stage of typhoid fever.	Explosive outbreak limited to 1 milk route.	Dr. F. E. Stone, Lynn, Mass. Special report.
16	September, 1906.	Kenton, Ohio	36	. . . .	28	Of the 36 cases, 28 were among patrons of 1 dairy which supplied only about 20 per cent of the milk used in Kenton. The other 8 cases were distributed among patrons of 2 different dairies and the users of milk from private cows. 5 milkmen had no cases at all on their routes. Cases of typhoid occurred at the dairy supplying the suspected milk. City and well water were excluded as a possible source.	Cases of typhoid were found at the milk farm.	. . . .	Preponderance of cases on 1 milk route.	Dr E. G. Horton. Ohio Sanitary Bulletin, July, 1906, p. 142.
17	August, 1906	Salt Lake City Utah.	29		29	Explosive outbreak. All cases were among patrons of 1 dairy. There was no common water supply. There had been a case of typhoid at the dairy the previous year and the excreta had been deposited in a privy without disinfection. Proper measures were taken to disinfect the privy vault and the outbreak came to an end	There had been a case on the dairy farm some months before.	. . . .	Explosive outbreak limited to patrons of 1 dairy.	Dr. T. B. Beatty, secretary State board of health, Salt Lake City, Utah. Special report.
18	August, 1906 . .	Providence R. I	18		18	Explosive outbreak limited to customers of 1 milkman supplied with can milk. Customers getting bottle milk were not affected	. . . .		Explosive outbreak limited to customers using can milk of 1 milk man	Dr. C. V. Chapin, superintendent of health, Providence. Report for 1906, p. 42.
19	August, 1906	Washington, D. C.	9		9	All circumstances pointed to milk as the carrier of infection 2 cases of typhoid were found in the family of the dairyman	On dairy farm .	Milk handled by persons coming in contact with sick.	Outbreak ceased when proper precautions were taken against the milk.	Dr. W. C. Woodward health officer, D. C. Special report
20	[illegible]	Trenton, N. J.	20		20	A case of "walking typhoid" was found at the dairy. Privy and well were close together	On dairy	Probably by polluted well water used in washing cans.	. . . . . .	Dr A. S. Fell, health officer, Trenton, N. J. Special report

MILK EPIDEMICS—Continued.

TABLE I.—*Typhoid*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
21	July, 1906.	Providence, R. I.	38	....	38	Outbreak explosive. All cases reported within a period of 2 weeks. 38 cases in 28 families. All took milk from 1 man, and all but 1 took bottled milk. According to laboratory standards the milk was one of the best in town. He had not been sterilizing his bottles, but began immediately to do so, and the epidemic rapidly subsided.	Probably a case in 1 of the families on the milk route	Probably bottles infected from case on milk route.	Explosive epidemic limited to customers of 1 milkman and ceasing when cans and bottles were properly sterilized.	Dr C. V. Chapin. In superintendent of health report of Providence for 1906, p 41.
22	July, 1906.	Washington D C.	23	... ...	23	All circumstances pointed to milk as the carrier of infection. It was found that an employee on the dairy farm had worked while suffering with typhoid	On dairy farm.	Milk handled by typhoid patient.	Outbreak ceased when proper precautions were taken against the milk	Dr W C Woodward, health officer, D C.
23	June, 1906.	Washington, D C Government Hospital for the Insane	5	1	5	5 attendants who had eaten ice cream at a small store where the wife and 2 children had typhoid at the time Came down synchronously with the disease. There were no other cases of the disease at the hospital. At the same time 8 members of the family and 2 employees of the dairyman who furnished milk to the storekeeper above mentioned had typhoid.	Presumably in shop selling ice cream.	Probably in making ice cream.	Explosive outbreak limited to persons having eaten ice cream at a shop where typhoid existed.	Dr Wm A White, Superintendent Government Hospital for the Insane, Washington, D. C. Special report.
24	May, 1906.	Charlotte, N. C.	17	... .	17	Explosive outbreak in girls' seminary. The milk supply up to Apr. 19 had been from	....	Probably from contaminated water	Relation of time of outbreak to change of milk	J. F. Anderson, passed assistant surgeon P H.

						cows kept at the school. From this date some milk was obtained regularly from a dairy. Between May 10 and 20, 17 cases of typhoid occurred. Persons using the same food and water supply but getting milk from other sources did not have typhoid. The water supply of the dairy was open to surface drainage and contained Colon bacilli as did also the public water supply.			supply; also fact that houses having same water but different milk were free from the disease.	and M. H. S. Public Health Reports 1906, XXI, Part II, p. 937.
25	April 1906	Scranton, Pa.	21	1	21	An unusual number of cases of typhoid were reported in one section of the city in April. Investigation showed that they were all customers of 1 milkman and that part of this man's milk came from a farm where there had been a case of typhoid late the previous autumn. Milk from this farm was excluded from sale and the epidemic stopped.	On farm producing milk.	....	Explosive outbreak limited to customers of 1 milkman. Outbreak ceased when sale of milk from infected farm was stopped.	Dr. W. E. Keller, superintendent bureau of health, Scranton, Pa. Special report.
26	April 1906	Providence, R. I.	14	..	14	Outbreak explosive. All were customers of 1 milkman on whose farm were found 2 cases of the disease, and 3 more cases developed later making 5 on farm and 14 in the city among customers. Sale of milk stopped and outbreak ceased.	Customers infected from milk contaminated probably by cases on farm. Cases on farm probably infected from original case, Mar. 26, in one of the customers.	Probably milk infected in handling on farm.	Explosive outbreak limited to customers of 1 milkman and stopping when sale of milk was abandoned. Finding of cases on farm.	Dr. C. V. Chapin, superintendent of health, Providence, R. I., in his annual report for 1906, p. 40.

MILK EPIDEMICS Continued.

TABLE I. *Typhoid* Continued.

No.	Date.	Place.	Number of cases	Number of deaths	Number of cases supplied with milk from the same dairy	Circumstances of outbreak	Location of original case or cases causing outbreak	Manner in which milk was infected.	Reasons for believing that milk carried the disease	Reporter and reference.
27	January, 1906	Charleston, S. C.	186	.....	155	In January and February, 1906, an unusual number of cases of typhoid were reported 186 during these 2 months. Of these, 132 were regular customers of a certain dairy A, and 21 more had used this milk away from home, 12 took no milk at all and 21 obtained it from various sources. The milk supply of Charleston is about 4,570 quarts daily, of which about 550 quarts are distributed by dairy A. Thus it will be seen that whereas dairy A handled only about one-eighth of the milk it had among the users of this milk over 82 per cent of the cases of typhoid. After the closing of the dairies the outbreak subsided. In December, 1905, there were reported 29 cases of typhoid, in January and February, 1906, 186 cases, in March, 1906, the number again fell to 27 cases.	Not found	Not known	Preponderance of cases on 1 milk route. Subsidence of outbreak 3 weeks after closure of dairy.	Dr. I. A. Ball and Dr. G. McF. Mood, Charleston, S. C. Special report.
28	1906	Brockton, Mass.	5	3	5	Unusual outbreak on 1 milk route. Case of typhoid at dairy.	At dairy		Outbreak on 1 milk route. Case of disease at dairy.	Geo. E. Bolling, Brockton, Mass. Special report.
29	1906	Baltimore	150	....	135(?)	Sudden development of many cases of typhoid on 1 milk	On farm producing milk in	By milker	Sudden outbreak on 1 milk	Dr. C. H. Jones, Assistant Com-

						route led to discovery that 1 of the farms supplying this milk had cases of the disease among the employees. Stopping sale of this milk put an end to the epidemic.	person or milker.		stopped when source of milk was changed.	Health, Baltimore, Md. From health department records.
30	October, 1905...	Port Deposit, Md.	19	1	19	Explosive outbreak limited to users of 1 milk supply where a case of typhoid was found on farm supplying milk. Sale of this milk was stopped and outbreak ceased.	On milk farm...	....	Explosive outbreak limited to consumers of milk from 1 dairy. Outbreak ceased when sale of milk was stopped.	Dr. H. L. Rich, Port Deposit, Md. Special report.
31	October, 1905..	Waltham, Mass...	86	......	43	Most of the cases occurred between Sept. 15 and Oct. 1. One-half of the cases were customers of 1 milkman. At 1 of the farms supplying milk to this dealer a boy of 12 years early in September took sick with what was diagnosed "walking typhoid."	Apparently in boy on dairy farm.	Apparently from boy on farm.	.	Mass. State Board of Health Report for 1905, p. 586.
32	September, 1905.	Washington, D. C..	16	......	16	All circumstances pointed to milk as carrier of disease. 8 cases were found in family on farm producing milk.	On dairy farm..	Same persons nursed sick, and handled milk.		Dr. W. C. Woodward, health officer D. C. Special report.
33	September, 1905.	Bedford, Mass	7	.	3	First 2 cases were employed by a farmer W. L. and boarders of F., a retail milk dealer. F. had milked for the farmer. 2 other cases occurred in families supplied with this milk. The dealer F. recovered Sept. 3 from a "slow fever." Another case was discovered in the family of a farmer supplying milk to dealer F.	The milk dealer F.	Apparently infection of milk.		Mass. State Board of Health Report for 1905, p. 580.
34	September, 1905.	Hartford, Conn.	50	4	50	Outbreak on 1 milk route. Cases limited to persons supplied with the night's milk. Night's milk was bottled and submerged in ice water. Morning's milk was not so treated. Shortly before outbreak dairy began using ice from sewage-contaminated pond for cooling night's milk. Ice was changed and new supply obtained and outbreak ceased.		Presumably by using sewage-contaminated ice in cooling tank in which bottled milk was submerged.	Sudden outbreak on 1 milk route limited to households supplied with night's milk.	Dr. C. P. Botsford, superintendent board of health Hartford, Conn. Special report.

MILK EPIDEMICS Continued.

TABLE I *Typhoid* Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
35	September, 1905	Hartford, Conn. .	24	3	24	Dairy used water from a spring-fed stream. About 2 weeks after a heavy rain a number of cases of typhoid appeared almost simultaneously on this milkman's route. The stream showed contamination, and the dairy was ordered to cease using the water. After about 2 weeks no new cases occurred on this route.	. .	Presumably from use of contaminated stream water.	Explosive outbreak on 1 milk route.	Dr. C. P. Botsford, superintendent board of health, Hartford, Conn. Special report.
36	August, 1905. . . .	Williamsport, Pa.	35	2	33	Cases were limited to the customers of 1 milkman, and almost every family supplied by him had 1 or more cases of the disease. Members of families using milk only in tea and coffee did not contract disease. Well on farm was found badly polluted by nearby privy. Milk was excluded from sale and in 2 weeks new cases ceased being reported.	Not known.	Probably by washing utensils in well water polluted from infected privy.	The limitation of cases to 1 milk route and the cessation of outbreak when milk was refused sale.	Dr. C. W. Youngman, health officer, Williamsport, Pa. Special report.
37	August, 1905. . . .	West Haven, Conn.	27	. . . .	23	In West Haven in 1905 typhoid was reported as follows: March, 1 case; June, 1 case, August, 28 cases. Of these 28 cases in August, 2 cases were late in the month and secondary to previous cases in same families, 1 case was of doubtful diagnosis. This leaves 25 primary cases which were			Explosive outbreak limited to and following 1 milk route.	Dr. C. D. Phelps. In Conn. State Board of Health Report 1905, p. 82.

						distributed in 24 families, of which 20 obtained milk from the same milkman, although there are a dozen or more large milk routes in West Haven. Precautions were taken against the milk and the last case was reported Aug. 26				
38	August, 1905	Woburn, Mass....	8	....	5	Five obtained milk from 1 dairyman, 1 was taken sick out of town. This dairyman obtained milk from a farmer 1 of whose employees had recently recovered from typhoid.	Employee of dairyman.	Apparently by handling of milk by infectious employee.	....	Mass. State Board of Health Report 1905, p. 588.
39	August, 1905.	Rockland, Mass..	15	......	15	15 cases typhoid were reported during latter half of August. All were traceable to milk coming from 1 dairy where the farmer had had typhoid and washed utensils in badly polluted well water.	Dairyman......	Apparently by washing cans with polluted water.	....	Mass. State Board of Health Report 1905, p. 584.
40	August, 1905	Swampscott, Mass.	14		11	11 of the cases were supplied by 1 dairyman who received milk from several farmers in the family 1 of which were found 2 cases of typhoid and 3 in another. At 1 of the farms a pail used for discharges of the patients was kept on the well curb, the water of which was used to wash cans.	In families of farmers supplying milk	Apparently from pollution of well water used to wash cans.		Mass. State Board of Health Report 1905, p. 586.
41	July, 1905	Westfield, Mass	38		22	22 of the cases obtained milk from 1 dealer who received milk from a farmer who had recently recovered from typhoid and also from another farmer whose wife had recently had typhoid. 2 of the cases had been acquired out of town.	Farmer of 1 farm and farmer's wife on another.	Probably direct in handling milk		Mass. State Board of Health Report 1905, p. 586.

MILK EPIDEMICS—Continued

Table I.—*Typhoid*—Continued

No.	Date	Place.	Number of cases	Number of deaths	Number of cases supplied with milk from the same dairy	Circumstances of outbreak	Location of original case or cases causing outbreak	Manner in which milk was infected	Reasons for believing that milk carried the disease	Reporter and reference
42	April, 1905....	Bowling Green, Ohio.	26		25	Outbreak explosive. 26 cases taken sick within 18 days. Water and schools were eliminated as sources of infection. 60 per cent of the cases were under 20 years of age. 25 of the cases used milk produced on 1 farm where the owner had had typhoid during the early part of the winter and the stools had been buried around the house and above the well. Thawing weather began March 10, typhoid epidemic began April 1.	Milker on dairy farm.	Probably by well water becoming polluted from discharges of typhoid patient which were buried in soil not far from well.	Explosive epidemic limited to 1 milk route.	Ohio State Board of Health Report 1905, p. 178.
43	January, 1905...	Rockland, Mass.	11	....	9	5 cases occurred in September, 1904, 1 case Oct. 1, 1 case Oct. 25, 1 case Jan. 8, 1905, 2 cases Jan. 10, and 1 case Jan. 15. 9 of the cases received milk from a dairy where all the work was done by father and son. The father was ill with typhoid Sept. 8, and returned to work Dec. 1.	Dairyman......	In handling milk.	....	Mass. State Board of Health Report 1905, p. 583.
44	1905	Brockton, Mass...	12	1	12	Unusual outbreak on route of 1 milkman. It was found that cans were washed with water from a well containing Colon baccilli and that typhoid dejecta had been thrown on ground not far from well. After closing this well no new cases occurred on this milk	On farm	Polluted water used in washing cans.	Outbreak on 1 milk route. Cases ceased when well on farm was closed.	Geo. E. Bolling, Brockton, Mass. Special report.

45	November, 1904.	Philadelphia, Pa. Twenty-sixth and Thirty-sixth wards.	55	....	35	Reported cases jumped from 3 to 21 cases in 1 week. Out of 55 cases 35 were supplied by milk from one dairy which received milk from a farm on which there had been 5 cases of typhoid in October and September. Nursing and milking was done by same persons. Chamber utensils washed at well with loose top Proper precautions were taken and after a week the number of notifications fell to normal.	On milk farm..	Same persons nursed sick and milked cows.	Sudden outbreak. Large number of cases with common milk supply. Finding typhoid on farm. Subsidence of outbreak when proper measures were enforced against the milk.	Report of Pa. State Board of Health, 1904-5, p. 270.
46	November, 1904.	Chester, Mass.....	9	1	9	All patients had obtained milk from the same farmer. The driver of the milk wagon had just recovered from typhoid.	Driver of milk wagon.	By driver.......	After dismissal of driver no new cases developed.	Mass. State Board of Health Report, 1905, p. 581.
47	October, 1904, to January, 1905.	Kolozsvár........	....	....	....	During October, November, and December, 1904, and January, 1905, the number of cases of typhoid reported were much above the average, so much so that an investigation was undertaken to find the cause The water could in no way be connected with the increase, and attention was attracted to a bake shop from which many cases seemed to originate A sample of milk taken from this shop was examined and the typhoid bacillus isolated from it Proper precautions were taken in regard to this shop and its milk, and the number of cases of typhoid fell in the next month back to the usual average number	....	....	....	Konradi Centralb. f. Bakt., etc., I Abt. Bd. 40, p. 31.
48	September, 1903	Govan........	20+	......	20	Majority of the cases were "directly and conclusively traced to the consumption of ice cream." The maker and vendor of the ice cream attended to his business for 2 weeks while ill with typhoid The outbreak was explosive	Maker and vendor of ice cream.	Making ice cream	Explosive outbreak among consumers of a certain ice cream	Doctor Barras, M. O. H. Pub. Health Lond., 1904-5, XVII, p. 224.

MILK EPIDEMICS—Continued.

TABLE I. *Typhoid* Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
49	September, 1904.	Rochester, N. Y.	13	......	13	Outbreak explosive. All cases reported between Sept 13 and Oct 17. Previously typhoid had been very rare in Rochester. Cases widely scattered in city. No social intercourse. Other sources of infection eliminated. Only thing in common was that all took milk from 1 farmer who had just nursed his wife through a case of typhoid and had also at same time handled the milk	In wife of dairyman.	By farmer nursing wife and also handling milk	Typhoid rare in Rochester. Explosive outbreak limited to 1 milk route and finding of case in wife of dairyman who also handled milk. Outbreak ceased when possibility of milk infection was removed	Dr Joseph Roby, Rochester, N. Y. Special report.
50	September, 1904.	Ipswich, Mass....	29	....	26	26 of the cases obtained milk from the same dealer, who supplied about 100 customers. Cans were washed in well water. Well badly polluted and near well was privy frequently used by passers by. These cases were all reported between Sept. 12 and Oct. 16.	Unknown	Apparently by washing cans in polluted well water.	The fact that so large a percentage of cases occurred on so small a milk route and the pollution of the well.	Mass. State Board of Health Report 1905, p. 582.
51	August, 1904....	Westchester, Pa..	45	....	33	Explosive outbreak. Of 45 cases 33 obtained milk from 1 dairy the milk of which came from 2 farms on which there was typhoid fever. Milk from these farms was stopped and the epidemic subsided.	On milk farms..	....	Explosive outbreak. Large percentage of cases on 1 milk route. Typhoid on farm, source of milk. Milk stopped and outbreak subsided	Report of Pa. State Board of Health, 1904–5, p. 9.
52	August, 1904...	Philadelphia, Pa.	78	....	41	Sudden increase from an aver-	At milk shop...	....	Large number of	Report of Pa.

		Twenty first Ward		1		age of from 1 to 3 cases a week to 15 cases. Out of 78 cases 41 took milk from 1 dairy and the other 37 were distributed among about a dozen dairymen. A visit to the suspected dairy showed the presence of typhoid. Proper measures were taken and in 4 weeks the occurrence of the disease had fallen to about normal where it remained.			cases with common milk supply. Finding typhoid at dairy. Subsidence of outbreak after instigation of proper measures. Elimination of water as cause. Explosiveness of outbreak.	State Board of Health, 1904-5, p. 269.
53	July, 1904.	Arad, Hungary	142	.	82	In the first half of the year there were 8 cases of typhoid in all in the town. Beginning July 19 there was an explosive outbreak. By Aug. 15 60 cases had been reported. The water was not at fault. It was ascertained that whipped cream coming from a house where there was typhoid fever was being sold at several confectionery shops and coffee houses and that 82 of the typhoid cases had eaten of this whipped cream previous to the onset of their illness. Of the 142 cases 67 were women and 33 children.	. . .	. . . .	. . . .	D. Konradi, Centralbl. f. Bakt., etc., I, Abt. Bd. XL, Heft 1, p. 35.
54	May, 1904.	Port Deposit, Md.	17	1	17	Explosive outbreak. All cases taken ill within a period of 30 days, and all had a common milk supply and were users of raw milk.	Previous to outbreak there had been a case on the farm diagnosed grippe.	. . . .	Unusual explosive outbreak occurring among users of a common milk supply.	Dr. H. L. Rich, Port Deposit, Md. Special report.
55	January, 1904	Quebec	25	1	25	Occurred at a seminary. Only certain of the pupils got milk to drink and all the cases were among the milk drinkers and nearly all the milk drinkers got typhoid. Three cases of typhoid were found in farm hands of a farm supplying milk to the seminary. The epidemic ceased when new milk supply was obtained.	On farm producing milk three farm hands had typhoid.	. . .	The epidemic ceased upon changing milk supply.	Dr. L. Catellier, Member Quebec provincial board of health, Quebec.

MILK EPIDEMICS Continued.

TABLE I. *Typhoid* Continued.

No	Date	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak	Location of original case or cases causing outbreak	Manner in which milk was infected	Reasons for believing that milk carried the disease.	Reporter and reference.
56	January, 1904.	Plymouth, Mass.	28	4	27	Town had previously been comparatively free from typhoid. 27 of the cases were supplied by the same milk dealer in whose family were found 5 cases of typhoid. Milking done by dealer's sons, 1 of whom worked 6 days after taking ill with typhoid Milk was handled in house.	Milk dealer's son who assisted at milking when ill with typhoid.	In milking	. . .	Mass. State Board of Health Report 1905, p 583
57	1904.	Milwaukee.	40	....	40	All cases bought milk coming from a farm on which cases of typhoid fever were found	On milk farm	. . .	All cases bought milk coming from infected farm.	Dr. A. W. Myers, Milwaukee, Wis.
58	1904.	McCloud, Cal.	30(?)	....	30(?)	Sharp epidemic The dairy was situated on the creek a short distance below the town. Creek received all the town sewage. It was claimed that the typhoid bacillus was found in the milk. Dairyman washed his cans by leaving them in the creek. Town's water supply was above suspicion coming from high up in the mountains.	....	Contaminated stream water presumably.	Sharp outbreak among users of certain milk	Dr. N. K. Foster Secretary Cal. State board of health. Special report
59	November, 1903.	Philadelphia, Pa	....		......	Sudden outbreak of typhoid in Twenty-sixth and Thirty-sixth wards; of 55 cases 35 took milk from 1 dealer, who it was found bought milk from a producer upon whose farm there had been 5 cases of typhoid during Sep-	On dairy farm, father and 4 children had the disease.	By mother nursing sick and milking cows.	Outbreak subsided when proper precautions were taken against the milk.	Dr. A. C. Abbott. Philadelphia Bureau of Health Report, 1903, p. 26.

						tember and early part of October.				
60	October, 1903	Los Angeles, Cal.	4	1	4	Milkman had typhoid. Supplied 40 families, among which 4 cases occurred. Dairy was closed.	Case on milk farm	. . . .	. . . .	Dr. L. M. Powers, health officer, Los Angeles, Cal.
61	September, 1903.	Fort Wingate, N. Mex	1	. . . .	1	A soldier arrived at the post and was put in charge of the company cow. On Aug. 31, 1903, he was admitted to hospital with typhoid, and one week later the only secondary case was admitted. He was in the habit of drinking all the milk he could get and very few other soldiers used this milk at all.	In milker	In milking and handling.	. . . .	Dr. F. F. Russell. Captain and assistant surgeon, U. S. Army.
62	August, 1903	Philadelphia, Pa.	78		41	In the Twenty-first Ward a localized epidemic became apparent about the end of August. Of the 78 cases, 41 obtained milk from 1 milk shop where there were 2 cases of typhoid.	There were 2 cases in house of milk seller.	Probably by handling of milk or by flies.	Predominance of cases on 1 milk route and fact that outbreak subsided when precautions were taken against milk. Other sources of infection were apparently eliminated.	Dr. A. C. Abbott. Philadelphia Bureau of Health Report, 1903, p. 23.
63	July, 1903.	Richmond, Va.			. . . .	Unusual outbreak on 1 milk route. The route of the wagon could be accurately plotted by the occurrence of typhoid cases.	. . . .	. . . .	Cases followed the milk wagon.	Dr. St. Geo. T. Grinnan, Richmond, Va. Special report.
64	April, 1903.	Palo Alto, Cal.			232	Out of 900 people supplied with milk from 1 dairy, 232 had typhoid. Of these, 216 were apparantly due to milk infection and 16 may have been due to secondary infection. All but 2 used the infected milk. The milk cans were washed and the milk diluted with water from a creek infected by sewage. Bacilli coli were isolated from the creek water and the milk.	In houses discharging sewage into creek.	Probably from contaminated water.	Cases limited to users of certain milk.	Modern Medicine Osler Vol. II, p. 85. Cited from Fish, Mosher, and Snow.

MILK EPIDEMICS—Continued.

TABLE I.—*Typhoid*—Continued.

No	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak	Location of original case or cases causing outbreak	Manner in which milk was infected	Reasons for believing that milk carried the disease.	Reporter and reference.
65	February, 1903..	Melrose, Mass....	7	......	6	From Feb. 28 to Mar. 22, 1903, 7 cases of typhoid were reported. 6 of these used the same milk. It was found that on 1 of the farms supplying milk to this dairyman there were 2 cases of typhoid. Milk from this farm was excluded from sale and no new cases were reported in Melrose during the next 3 months.	On dairy farm.	In handling milk in all probability.	Cases practically limited to 1 milkman. The finding of typhoid on dairy farm. The cessation of outbreak when milk from infected farm was excluded from sale.	Dr. C. P. Holden, Chairman of board of health, Melrose, Mass.
66	1903............	Baltimore, Md....	26	4	26	26 cases of typhoid developed in rapid succession among employees of 1 factory. Factory served dairy lunch, the milk of which came from a farm where the water supply was found polluted with Colon bacilli.	.	..	Epidemic ceased when milk supply was changed.	Health department records, Baltimore, Md. C. Hampson Jones, M. D. Assistant commissioner of health.
67	December, 1902.	Montclair and Bloomfield, N.J.	28	....	28	All cases took milk from 1 dairy and were limited to those getting pint bottles. After much investigation it was found that at 1 house taking daily 3 pint bottles of milk a case of typhoid had existed for a few days until he could be sent to a hospital. This case had come from New York and had not been reported. The dairy in question did not sterilize its bottles and pint bottles used in the house with typhoid would	In family on milk route.	By infected bottles being refilled with milk without previous sterilization.	The epidemic ceased when dairy was closed.	Montclair Board of Health, Ninth Annual Report, 1903, p. 29.

						be washed, filled with milk, and delivered to other customers. The dairy was closed and in 2 weeks new cases ceased to be reported.				
68	October, 1902	Washington, D.C.	14		14	All circumstances pointed to milk as carrier of infection. There had been a case of typhoid on farm supplying milk and the excreta were thrown on ground around well.	Case on dairy farm.	By washing cans in water from infected well.	Outbreak ceased after proper precautions were taken against dairy.	Dr. W. C. Woodward, health officer, D. C.
69	September, 1902	Belmont, Mass.	6		6	Outbreak explosive, 6 cases reported in 1 week. All obtained milk from 1 man who supplied only a few families and who had a few months previously had typhoid. He washed milk utensils with water from a dug well near house. Well showed pollution.	Probably on milk farm.	Probably by washing milk cans with polluted water.	Explosive outbreak limited to 1 milk route.	Dr. F. L. Morse, Mass. State Board of Health Report 1902, p. 567.
70	August, 1902	Washington, D.C.	15		15	4 cases of typhoid were found on dairy farm. Circumstances pointed conclusively to milk as carrier of infection.	On dairy farm.	Same persons nursed sick and handled milk.	Outbreak ceased when proper action was taken against dairy.	Dr. W. C. Woodward, health officer, D. C.
71	July, 1902	Washington, D.C.	6		6	All circumstances pointed to milk as the carrier of the infection and a case of typhoid was found on the dairy farm.	On dairy farm.	Persons handling milk also nursed patient.	Outbreak ceased when shipment of milk was stopped.	Dr. W. C. Woodward, health officer, D. C.
72	January to March 1902	Niles, Ohio	107		63	Out of 107 cases 63 were on 1 milk route. There were in Niles 5 other milk routes as large as this one, but among the 5 there were only 34 cases. There was a case of typhoid on the farm.	There was a case of typhoid on the farm.		The excessive number of cases on 1 milk route.	Dr. E. G. Horton, Seventeenth Annual Report, Ohio State Board of Health, 1902.
73	October, 1901	Clydach, Glamorganshire	13		13	Explosive outbreak between Oct. 18 and 30 in town of 2,000 population. Man on milk farm had been feeling unwell from Sept. 22, but continued at work. On Oct. 12 his illness was diagnosed typhoid and he died Oct. 17. On Oct. 12 the handling of the milk was removed entirely from the house. All cases in town developed between Oct. 18 and 30 and were consumers of milk from the infected farm. There had been no typhoid in Clydach during the preceding 26 months.	On milk farm.	By milker	Explosive outbreak limited to consumers of 1 milk supply. The original case found on milk farm. The cessation of outbreak 18 days after dairy was removed from the farm.	W. Williams, M. A., M. D., M. O. H., Pub. Health, Lond., 1901-2, XIV, p. 650.

MILK EPIDEMICS—Continued.

TABLE I. *Typhoid*—Continued.

No	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference
74	September, 1901.	Nahant, Mass....	12	.	12	Outbreak explosive. 12 cases of typhoid reported in 10 days. All had a common milk supply. Investigation showed that the farm producing the milk used badly polluted well water to wash the cans. No case of the disease was found on the farm.	.....	......	Explosive outbreak limited to 1 milk route	Dr. F. L. Morse. Mass. State Board of Health Report 1901, p. 580.
75	September, 1901	Wallasey, Cheshire.	90	......	50	Sanitary defects at farm producing milk. Water contained many B. coli. Stoppage of milk stopped outbreak.		Probably by contaminated water.	. . .	Dr. A. Craigmile. Annual Report M. O. H. Wallasey 1901. Cited by Swithinbank and Newman.
76	August, 1901..	Buffalo, N. Y.	21		21	Outbreak of typhoid on milk route. There had been a case of the disease at the dairy 9 months previously.	Supposed to have possibly been due to a case occurring at dairy 9 months before.	..	An unusual number of cases on 1 milk route	Dr. Ernest Wende, Buffalo, N. Y. Special report.
77	August, 1901	Quebec...	40	15	40	Quebec is practically free from typhoid. 40 cases appeared at 1 nunnery, consisting of 3 buildings. All cases occurred in 1 building which had a separate milk supply. 2 farm hands on the farm supplying milk were found to have recently had typhoid. When a different milk supply was obtained cases ceased to develop.	On the milk farm 2 farm hands.	......	When milk supply was changed epidemic ceased	Dr. L. Catellier. Member Quebec provincial board of health. Quebec

78	July, 1901..	Wallasey, Cheshire.	26		24	Grave sanitary defects found at farm producing milk. Water supply poor. Bacillus coli commune found in milk.	....	....		Dr. A. Craigmile. Annual Report M. O. H. Wallasey, 1901 Cited by Swithinbank and Newman.
79	February, 1901..	Spring Grove, Pa.	28	3	27	Town population, 1,000. Explosive outbreak, 28 cases reported in February. The first 15 cases were reported within 4 days. All families invaded but 1 used milk from 1 dairy the milk of which came from a farm where 3 cases of typhoid existed antedating the outbreak in town. On this farm the dejecta were thrown on the ground around the well the water of which was used to wash the milk cans. The family also nursed the sick and milked the cows.	3 cases on dairy farm.	Dejecta thrown on ground around well, also by nurses milking cows.	Explosive outbreak limited to consumers of milk from 1 dairy and cessation of outbreak after closure of dairy.	Dr. J. C. Gable, Pa. Med. Jour. 1902-3, VI, p. 145.
80	1901	Melbourne	6		6	Dairyman ill with typhoid at hospital from Dec. 6 to Jan. 15. Did not go to work till latter part of February. He supplied 154 houses with scalded milk and 22 with raw milk. In the latter 22 houses 5 cases of typhoid developed between Mar. 4 and 19. The dairyman was isolated and outbreak ended. This dairyman was considered to have been a typhoid bacillus carrier, and to have thus infected the milk	Dairyman......	Probably in handling milk.	Cases limited to consumers of milk from dairy where keeper had just recovered from typhoid.	Dr. James Jamieson, Pub. Health Lond., 1901-2, XIV, p. 655.
81	1901.	Ware, Mass	20			For several years past Ware had had only 1 or 2 cases of typhoid during the year. In 1901 20 cases occurred and were traced to an infected milk supply, and when this was cut off the epidemic rapidly abated	....	....	.. ..	Mass. State Board of Health Report, 1901, p. 589.
82	1901.	Los Angeles	8		8	Milkman had typhoid. Proper precautions were taken and cases soon ceased.	Case on dairy farm.	....	. .	Dr. L. M. Powers, health officer, Los Angeles, Cal.

MILK EPIDEMICS Continued.

TABLE 1. *Typhoid* Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
83	November, 1900.	Waltham, Mass.	7		7	Outbreak explosive. All obtained milk from 1 man, A. Some of A's milk was obtained from a farm where there had been a case of typhoid in August. The patient's dejecta entered a cesspool which had been cleaned recently and its contents spread on ground around it. The man who cleaned the cesspool also milked the cows.	On milk farm.	Probably by milker cleaning cesspool and then milking after an insufficient toilet	Epidemic ceased when proper precautions were enforced at milk farm	Dr. F. L. Morse. Mass. State Board of Health Report, 1900, p. 854.
84	November, 1900.	Salford	27	......	18	Dairyman was ill with probable typhoid fever.	Probably at dairy.	....	......	Dr. Niven, M. O. H. Annual Report, 1900, p. 79. Cited by Swithinbank and Newman.
85	October, 1900.	Beverly and Salem, Mass.	26	...	22	Outbreak explosive. 22 got milk from common source, 1 evidently contracted disease out of town, 3 gave no definite data. The dairy supplying the milk was inspected and farmer's son found ill with typhoid. He also obtained some milk from farmer B, on whose farm were found 7 cases of typhoid. Two of these cases were milkers.	On dairy farms supplying milk.	By handling or possibly by washing cans with polluted spring water.	Epidemic ceased when sale of milk was stopped.	Dr. F. L. Morse. Mass. State Board of Health Report 1900, p. 803.
86	October, 1900	Elkton, Md.	64	2	64	Wife of dairyman nursed a neighbor with typhoid and became ill herself. Son also	A neighbor's wife nursed by wife of	By family of dairyman.	Explosive outbreak limited to houses sup-	Dr. J. S. Fulton. Jour. of Hyg. Lond., I, p. 4[illegible]

						became ill. Following this typhoid appeared in Elkton in houses supplied by this dairy. The outbreak was explosive in its incidence. Of the 39 families invaded all got milk from this dairy. They used various water supplies, 21 used town water and 18 private wells.	dairyman. Dairyman's wife became infected and ill as well as his son.		plied by milk from infected dairy	
87	October, 1900...	Iowa State College of Agriculture and Mechanic Arts, Ames, Iowa.	65	2+	65	College milk ordinarily received from 2 farmers—Sketon and Pritchard. From Sept. 3 to Oct. 17 Farmer Briley also furnished milk. Briley's daughter took ill with typhoid Aug. 3. The first cases in the college appeared Oct. 8. All cases of typhoid at the college occurred among milk drinkers. Of the 16 football players who ate at the training table and were encouraged to drink much milk, 13 contracted typhoid. Briley's milk was discontinued Oct. 17 and the last case fell ill 17 days later. Water from 45-foot well on Briley's farm showed gross contamination and an organism was isolated from the water resembling, if not identical with, the Eberth bacillus. This water was used to wash milk cans which were not scalded. All persons attacked were milk drinkers.	At milk farm. .	Probably through polluted water used in washing cans.	Outbreak limited entirely to users of a certain milk and its cessation when milk was stopped.	Dr. W. E. Harriman. Iowa State Board of Health Report 1901, p. 101.
88	September, 1900	Cheyenne, Wyo. .	300+	20(?)	300(?)	Wife of dairyman had typhoid fever. A little later other cases developed at the dairy. New cases appeared throughout the city. All the early cases were among consumers of milk from this dairy	At dairy farm. . .	. . . .	Sudden outbreak among patrons of 1 dairy at a time when there was little if any typhoid in the city	Dr. G. P. Johnston, Cheyenne, Wyo. Special report.

MILK EPIDEMICS Continued.

TABLE I. *Typhoid* Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
89	September, 1900.	Franklin, Mass.	23	...	8 of A 9 of B	Epidemic was somewhat explosive. 8 cases took milk from A whose daughter had typhoid. Farmer B had but 1 cow and supplied only 3 families and in these 3 families there were 9 cases of the disease. B's wife had typhoid and 2 of his children later contracted it. This would seem to be really 2 small epidemics.	In case of A the daughter did not live on farm, but probably infected milk cans. In case of B the case was that of his wife on the farm	In A probably by infection of cans. In B the same. Conditions being very unsanitary in house and barn	. .	Dr. F. L. Morse. Mass. State Board of Health Report, 1900. p. 825.
90	September, 1900.	North Adams, Mass.	30	..	10	Of the first 30 cases reported 10 obtained milk from one man, A, 4 from another, B, and 5 from various supplies. Of the other 11, 9 probably contracted it out of town, 1, a nurse, had nursed a case, and 1 gave no data. On investigation A's son was found to have taken ill with typhoid Aug. 15, and was nursed by his mother, who also did the housework. All the cases on A's route developed at such a time that they could have been infected from his son. B's water supply was found polluted.	On milk farm..	Probably cans infected in handling and cleansing	The possibility of infection, the time it occurred, and the fact that correction of conditions at farms was followed by a marked subsidence of the epidemic.	Dr. F. L. Morse. Mass. State Board of Health Report 1900, p. 843.
91	July, 1900..	Norwood, Mass...	45	....	45	All 45 cases were reported within 34 days and all obtained	Unknown.. . .	From water of polluted well	Epidemic ceased when C was for-	Dr. F. L. Morse. Mass. State

						milk from A. A obtained milk from Farmer C who used for washing milk cans water from a polluted well which had been the undoubted source of a similar epidemic 6 months previously.		used in washing cans.	bidden to sell milk, only 2 cases being reported in the next 2 months.	Board of Health; Report 1900, p 848.
92	July, 1900.	Glasgow	34	....	34	Milk was only thing common to all cases. Relatives of farmer had had typhoid. Dairy farm water supply was open to criticism.	....	....	.. ..	Report of M. O. H. of Glasgow, 1899–1900, p. 45. Cited by Swithinbank and Newman.
93	June, 1900.	Hamar, Norway	53	......	42	No typhoid in Hamar since 1896. In 1900 an explosive outbreak occurred. Of the 53 cases 42 drank milk from shop where the owner and his wife had typhoid, the latter tending the shop while still ill, 6 of the cases were contracted by personal contact and 3 indirectly.	Milk vendor in milk shop.	Probably in handling with soiled hands.	Explosive epidemic. Original case in milk shop. Most of other cases among the customers of this shop.	Heme, Tidsskrift f. den norske Lageforening. Kristiania, July, 1901. Abstract Pub. Health. Lond. 1901 2. XIV, p. 403.
94	January, 1900.	Nashville, Tenn.	20	2	20	Typhoid invaded nearly every family supplied by the suspected dairy and those which escaped were not in the habit of drinking milk. 2 cases were found at dairy. Dairy well was shallow	Cases at farm	. . . . ..	Cases limited largely to users of milk from 1 dairy	S. E. Cox, M. D., county health officer. Special report
95	January, 1900.	Norwood, Mass.	10	. . .	10	10 cases were reported in a little over a month. All obtained milk from A. The water supply of 1 of the farms supplying milk to A was found polluted. The water supply was changed and in the next month no cases were reported.	Unknown.	From water of a polluted well which was used in washing cans.	The epidemic ceased when milk from this farm was refused sale, no cases being reported in the next month.	Dr. F. L. Morse, Mass. State Board of Health Report 1900, p 846.
96	January 1900	Brockton, Mass.	21	. . . .	21	All obtained milk from same source. Nothing else in common. Cases developed between Nov. 27, 1899, and Feb. 7, 1900. At dairy polluted water was used to wash cans. No case of typhoid itself was found	Not known.	By washing cans in polluted water.	When sale of milk from this farm was stopped the epidemic ceased.	Dr. F. L. Morse, Mass. State Board of Health Report 1900, p. 815.

MILK EPIDEMICS—Continued.

Table I *Typhoid* Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
97	1900... .	Milton, Mass.. ..	7	.. ..	7	There were 7 cases in all. These occurred over a period of several months. All were supplied with milk from Farmer A who had but 2 cows and supplied but a few families. No other cases of typhoid were present in this part of town A's water supply was found badly polluted.	Not known.	Probably by washing cans with polluted water.	No new cases developed after correction of water supply on farm.	Dr. F. L. Morse. Mass. State Board of Health Report 1900. p 841.
98	November, 1899.	York. Pa........	82	14	66	Explosive outbreak Not evenly scattered throughout town, but limited to certain sections Although 50 milk dealers have routes in York, over 80 per cent of the cases were customers of 1 milkman. Some of the other cases may also have used this milk, as this man occasionally sold milk to other dealers. 5 of the cases were imported. This suspected milk, it was found, came from a farm where there had been 5 cases of "typho-malarial" fever, 1 of which had died. The dejecta had been thrown without disinfection on the ground within a few feet of the spring where the milk cans and pails were washed Dairy was closed, and in 2 weeks the outbreak was at an end.	5 cases on dairy farm.	By throwing dejecta around spring where milk cans were washed.	Explosive outbreak limited almost entirely to users of milk from 1 dairy and its cessation 14 days after dairy was closed.	Dr. I. C. Gable. Pa. Med. Jour., 1902-3, VI. 143.

99	October, 1899...	Fairhaven, Mass.	21	....	16	Outbreak explosive. 16 became ill between Sept. 10 and Oct. 6, and all obtained milk from 1 source where the milking was done by a man who had had a severe attack of typhoid some months previously. The other 5 cases were supposed to have some other origin.	Apparently on dairy farm.	Apparently by a bacillus carrier doing the milking.	Explosive outbreak. Common milk supply probability of bacillus carrier doing milking.	Dr. F. L. Morse. Mass. State Board of Health Report 1899, p. 740.
100	September, 1899.	Boston (Dorchester), Mass.	57	....	48	Outbreak explosive, between Sept. 29 and Oct. 13. Of the 57 cases in Dorchester, 48 took milk from 1 milkman. A case of typhoid was found in the family of a farmer producing part of the milk delivered by this milkman.	In family of farmer producing milk.	Father of patient took care of stools and also milked cows.	Explosive outbreak limited to 1 milk route, and case of typhoid found on farm at a time possible to have caused outbreak.	Dr. Burr. Jour. Mass. Assoc. of Boards of Health, Jan., 1900, p. 142.
101	August, 1899....	Dublin	66	....	66	There were cases of typhoid at the dairy, which was small and sold the milk of 18 cows. Wherever this milk was used typhoid appeared, and in several institutions using it, only those consuming this milk were attacked. The clear cut onset of the disease in persons using it and the freedom from attack of those using other milk was clearly demonstrated in several instances.	At dairy	Probably by nurse of sick handling milk.	Sudden outbreak among consumers of milk of 1 dairy and freedom from attack of others.	Sir C. A. Cameron. Dublin Jour. Med. Sc. 1899, p. 330.
102	August, 1899.	Stoneham, Mass..	25	.	20	Outbreak was explosive. 20 of the cases got milk from common source and some of the other 5 may have drunk some of this milk. The first case appeared on this route July 17. One of the drivers became ill Aug. 17.	First on milk route, later at dairy.	Apparently infection of bottles and cans by typhoid case on the milk route.	Common milk supply of cases. Epidemic ceased when original case, which was supposed to be source of infection, died.	Dr. F. L. Morse. Mass. State Board of Health Report 1899, p. 759.
103	June, 1899.	Bullingen and Hunningen and Murringen	27	1	23	Explosive outbreak among patrons of 1 dairy. Cases were scattered. There had been in May typhoid in 2 families supplying milk to this dairy. The milk was ordered pasteurized and outbreak came to an end.	In families supplying milk to dairy.	....	Explosive outbreaks practically limited to customers of 1 dairy and cessation of outbreak when proper precautions were taken.	Dr. Schlegtendal. Vierteljahrsschr. f. offentl. Gesundheitspflege. 1900, XXXII, p. 298.

MILK EPIDEMICS—Continued

TABLE I—*Typhoid*—Continued

No.	Date.	Place	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy	Circumstances of outbreak	Location of original case or cases causing outbreak	Manner in which milk was infected.	Reasons for believing that milk carried the disease	Reporter and reference
104	April, 1899	Chelsea and Boston, Mass.	78	......	60	Outbreak explosive 60 of the 78 cases obtained milk from same source. No other source of infection was found. There was found at the milk depot a man who had had a severe attack of typhoid and returned to work Mar 20 and handled the milk.	Apparently at distributing depot	Apparently by handling milk.	Typhoid appeared wherever this dairy supplied milk	Dr. F. L. Morse Mass State Board of Health Report, 1899, p 737
105	March, 1899	Andover, Mass...	11	....	10	Outbreak was explosive 11 cases reported between Mar. 20 and Apr. 4. 10 of the cases used milk from the same source and the proprietor of this dairy was found to have been ill from Mar. 10 to 30. Working on this knowledge the epidemic ceased	Proprietor of small dairy farm	Milking and handling milk	Outbreak ceased when precautions were taken to prevent contamination of milk	Dr. F L Morse Mass. State Board of Health Report 1899, p 730
106	1899	Paisley.......	44	......	44	Explosive outbreak Limited apparently to users of milk from 1 dairy where there had been a succession of 3 cases of typhoid Dairy was closed and outbreak ceased.	At dairy........	Probably by nurses also milking cows.	Explosive outbreak among users of milk from an infected dairy and cessation of outbreak when dairy was closed.	Dr. Wm. Robertson, M. O. H Annual Report 1899 Abstr. San Jour. Glasgow 1900 1. VIi, p 308
107	December, 1898..	New York City, N. Y.	17	......	13	"In district bounded by Mott and Walton avenues and 138th and 152d streets, 17 cases of typhoid occurred. All but 4 were traced to in	....	....	......	Med News 1898, LXXIII, p. 800 Cited by Dr G M. Kober

						fected milk sold by grocer in neighborhood."				
108	November, 1898.	Glasgow.	43	1	43	The 43 cases occurred within 2 weeks. Typhoid in family of girl working in milkshop. This girl contracted it also. Milk from same source and delivered by same driver but which was not handled at the dairy where the girl worked did not produce typhoid among consumers. The cases were limited to users of milk handled by girl	In family of shopgirl	Probably in handling.		Dr. A. K. Chalmers. M. O. H Report 1898. Cited by Swithinbank and Newman.
109	July, 1898.	Strauch (Kreis Montjoie).	3		3	Son returned home to farm sick Dejecta were thrown into the garden. In from 2 to 4 weeks father, mother, and 2 sisters took sick with typhoid so mild that they continued for a time to milk the cows and handle the milk. This milk was taken to a dairy. By the middle of August, 3 cases developed in the town in different houses, widely separated and with nothing in common All usual means of infection were eliminated It was found that the 3 had drunk at the dairy milk coming from this infected farm	On milk farm.	Infected persons milking.		Dr. Schlegtendal. Vierteljahrsschr. f. öffentl Gesundheitspflege 1900, XXXII, p. 298.
110	February, 1898.	Lascheid.	73	10		A dairy association had been formed. In the beginning of February there was a case of typhoid in the town At the end of the month many cases began to be reported 73 cases in 26 households 21 of these families were patrons of the dairy Communities having members in the dairy association had typhoid the others did not.			Great preponderance of cases among patrons of dairy association.	Dr Schlegtendal. Vierteljahrsschr. f öffentl Gesundheitspflege 1900, XXXII, p. 298
111	January 1898	Paterson, N. J.	27		19	19 of cases were supplied with milk from dairy where milkman was sick with typhoid and had in the early stages been engaged in handling the milk.	At Dairy			Dr J L Leal Trans Med. Soc N J 1898, p 176 Cited by Dr G M Kober.

MILK EPIDEMICS—Continued.

Table I.—*Typhoid*—Continued.

No	Date	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference
112	1898.	Lubeck suburb of.	25	...	25	All cases had obtained milk from 1 source where child of milk dealer had typhoid. Evidence pointed to fact that infection had been carried by washing milk cans in water infected with child's dejecta.	Child of milk dealer	...	Outbreak limited to consumers of milk from one dairy	Riedel, Zeitschft. Med.-Beamt. Heft 3, 1898. Cited by Dr. G. M. Kober.
113	November, 1897.	Paterson, N. J.	142		137	All but 5 cases received milk from same creamery, where 3 sons of proprietor were or had been suffering from typhoid. The drain from premises occupied by patients reached a stream and emptied within 15 feet from a spring. Water from both sources had been used for washing utensils. Eberths' bacillus claimed to have been isolated in the water. The patients attended to dairy business during early stages of disease.	At Creamery....	....	...	Dr. J. L. Leal. Trans. Med. Soc. N. J. 1898, p. 176. Cited by Dr. G. M. Kober.
114	October, 1897	Liverpool.	27		27	Explosive outbreak of typhoid among children all of whom had eaten ice cream or chipped potatoes at the stand of an Italian vendor who had a case of typhoid in his family at the time. There was nothing else in common. Some lived in various parts of the city and attended different schools.	Wife of vendor of ice cream.	Probably in manufacture or handling of ice cream.	Explosive outbreak among children who had eaten ice cream at a certain vendor's stand.	Dr. E. W. Hope. Liverpool Med. Chir. Jour. 1898, XVIII, p. 185.

115	October, 1897..	Clifton, England..	244	....	230	Clifton had population of 47,553. Outbreak explosive. Practically no typhoid in Clifton when outbreak occurred. On 1 milk route (A) 56 houses were supplied of which 31 had typhoid. Of 453 persons in these houses 114 contracted typhoid. Another man (B) selling this same milk, produced by X, had a route of 40 houses of which 18 were invaded with 28 cases of typhoid. 230 of the 244 cases were shown to have used this milk produced at the suspected dairy.	Not found . .	Probably by polluted brook water.	Its limitation to persons using milk from 1 source and its subsidence when this milk ceased to be sold.	Dr. D. S. Davies M. O. H. Bristol Trans. Epidem. Soc. London, 1897-8, p. 78.
116	September 1896.	County of Cork...	35	......	35	All patients had used separated milk from a creamery at Liscarroll to which milk from an infected house had been sent during illness of typhoid patient.	At farm sending milk to creamery.	. . . .	Outbreak limited to patrons of 1 creamery.	Dr. Browne. Lancet, Lond., 1896, II, 1500. Cited by Dr G. M. Kober
117	Autumn, 1897...	Univ. of Va.......	14	....	14	Cases occurred on 1 milk route. The proprietor was also nursing a neighbor ill with typhoid.	Near dairy.....	Milkman also acted as nurse	Cases occurred on 1 milk route.	Dr. J. S. Davis Univ. of Va. Special report
118	July, 1897.	Lubeck..........	22	....	18	18 of the 22 cases obtained milk from 1 farm. All used raw milk. Of 23 families regularly supplied by this farm 12 were invaded by typhoid. When proper measures were taken the outbreak ceased. There had been a case of typhoid on this farm during the latter part of June, and the well where the milk cans were washed had undoubtedly become infected	On farm.....	From water used to wash milk utensils	Sudden outbreak almost entirely limited to houses supplied with milk from one farm. Case on farm. Protection against milk stopped outbreak.	Riedel Zeitschr f. Med.-Beamte 1898, 74. Cited by Dr Schlegtendal.
119	May, 1896..	Kirkcaldy...	191	20	Nearly all.	Scarcely a house in which the suspected milk was used escaped typhoid. The epidemic ceased after closure of the dairy. First case was reported May 17. It and subsequent cases were shown to have arisen from this dairy where an employee had typhoid.	At dairy farm...		The coincidence of the use of the milk and invasion of the disease and cessation of outbreak when dairy was closed.	Dr. Mackay, M. O. H. Cowkeeper and Dairyman's Journal Oct. 1896. Cited by Hart also Brit M. J. 1896, II, 694.

MILK EPIDEMICS—Continued.

TABLE 1. *Typhoid*—Continued.

No.	Date	Place	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference
120	May, 1896	Dublin . . .	14		14	2 children of owner of milk shop had typhoid. The business was very small and yet 14 cases of typhoid were found among the customers. The living apartments and shop were closely connected	Children of owner of milk shop.	Probably by nurse of children handling milk	. . .	Sir C. A. Cameron, M.O.H. Dublin. Brit. M. J., 1896, II, 441.
121	March, 1899	Buffalo, N. Y . .	14	. .	14	Rapid sequence of 14 cases found on 1 milk route. It was found that several months previously a case of typhoid had existed in the house where this milkman lived and handled his milk. Conditions were insanitary. Sale of milk was stopped and outbreak ceased	Probably in house occupied by milkman and his milk room.	. . . .	Rapid sequence of cases on 1 milk route and its cessation when dairy was closed	Dr. E. Wende, Jour. Am. Med. Ass., 1900, XXXIV p. 151.
122	1898	Stadt Aachen				In a part of the town supplied with good water there occurred an unusual number of typhoid cases. Their common milk supply was traced to a farm where there had been several cases of typhoid. The well where the milk utensils were washed had become infected. When the sale of milk was stopped the epidemic ceased.	On milk farm . .	By washing milk utensils in infected water.	Outbreak limited to customers of a certain milkman, and its cessation when sale of milk was stopped.	Dr. Schlegtendal. Vierteljahrsschr. f. öffentl. Gesundheitspflege, 1900, XXXII, p. 290.
123	1896. . . .	Strassburg, i. E				"Dr. Sommerfeld reports epidemic of typhoid in barracks, which was traced to specifically infected water with which utensils were washed	....	....	....	Dr. P. Sommerfeld Heilkunde, Wien, 1896-7, I, p. 167. Cited by Dr. G. M. Kober.

					and the milk adulterated; he also referred to smaller epidemic reported by Rehn in which the latter demonstrated in the milk the presence of the bact. Coli commune."				
124	November, 1895.	St. Helens, Lancs.	12	2	12	All cases users of 1 milk supply. Epidemic ceased when dairy was closed. Case of typhoid on farm. Of 37 families supplied 8 were invaded.	....	. ..	Cases limited to users of 1 milk supply and outbreak ceased when dairy closed.	Dr. Robertson, M. O. H. Special report of M. O. H. Cited by Hart.
125	November, 1895.	Providence, R. I.	31	3	31	Outbreak explosive, all obtained milk from same dairy, on which were found 2 cases of typhoid. The mother nursed the cases and washed the milk utensils and cloth used for straining. There had been no typhoid prevailing in city previously.	On milk farm...	By same persons nursing cases and washing milk utensils.	Epidemic ceased after removing milk of this farm from sale. Typhoid had not prevailed previously in the city.	Dr. Charles V. Chapin, Supt. of Health, Providence, R. I., in 1895 Annual Report.
126	August, 1895.	Fintry, England..	22	.	22	Population of Fintry, 300. Had no typhoid since 1878. Outbreak explosive in August, 22 cases in all; 17 were primary cases among milk consumers of 1 cow. The other 5 cases were considered secondary cases in infected houses. Milk of cow was used in 17 families, of which 13 were invaded and of the 65 persons in these families 22 fell ill.	....	... .	.. . . . .	J. C. McVail, Brit M. J., 1896, II. 217
127	August, 1897.	Buffalo, N. Y. ..	18	. .	18	Rapid outbreak limited to 1 milk route. Investigation revealed that the wife of one of the dairy employees had typhoid and that her husband acted as nurse at night and as dairyman during the day. Proper precautions were taken and the outbreak ceased	Wife of dairy employee.	Dairy employee acted as nurse at night and dairyman during day.	Outbreak limited to customers of 1 dairy connected with which was found a case of typhoid. Cessation of outbreak upon isolation of original case.	Dr. E. Wende Jour. Am. Med Assoc., 1900. XXXIV, p. 151

MILK EPIDEMICS—Continued.

TABLE I.—*Typhoid*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
28	May, 1895	Plumstead.	177	23	159	Outbreak explosive. 90 per cent of cases were users of milk from 1 dairy. In 1 road of 49 houses supplied by this milk 29 were invaded while of 150 other houses in the same neighborhood but using other milk only 2 were invaded. Well water on farm was polluted. Epidemic declined rapidly 2 weeks after closure of dairy. Total number of families supplied by milkman 250.			Explosive outbreak following and limited to 1 milk route and ceasing when dairy was closed.	Dr. S. Davies, M. O. H. Pub. Health, Jan., 1896. Cited by Hart.
29	189[illegible]	Warminster, Wilts.	47	3	34	Of the 47 cases, 34 were users of the suspected milk, 11 were constantly associated with these users, and 2 were imported. On farm producing milk a sewage-polluted brook ran through pasture where cows grazed and were milked.		Cows grazed and were milked in pasture through which ran a sewage-polluted brook.	Practical limitation of cases to 1 milk route.	Dr. Flower, M. O. H. Special Report of M. O. H. Cited by Hart.
30	October, 1894	Titusville, Pa.	50	4	46	A person infected with typhoid in Buffalo was taken sick on dairy farm near Titusville, Pa. 2 more cases developed on farm. The mother took care of the sick and also the milk. Within 4 weeks there were 46 cases of typhoid in families using milk from this farm. The 4 other cases had been out of town and had apparently become infected	On dairy farm.	Same person nursing sick and handling milk.	Explosive outbreak limited to customers of 1 milkman, and finding same person on farm nursing sick and handling milk.	W. G. Johnston, Rep. Pa. State Board of Health 1894, p. 258.

						while away. Of the 46 cases 4 became ill later than 2 weeks after closure of dairy, but they occurred in families already infected and most likely were secondary and infected by existing cases in the families			
131	September, 1894.	Buffalo, N. Y.....	19		19	Explosive outbreak among customers of 1 milkman whose wife was found convalescing from typhoid. The husband nursed his wife, who was in a room adjoining the milk room, and handled milk and washed the cans. Dairy was closed and outbreak ceased.	Wife of milkman.	Milkman nursed wife and handled milk and washed cans.	Explosive outbreak limited to customers of 1 milkman whose wife was ill at the time and cessation of outbreak when sale of milk was stopped.	Dr. E. Wende. Jour. A. M. A., 1900, XXXIV, p. 151
132	September, 1894.	Marlboro, Mass.	49	7	49	All cases occurred between Aug. 1 and Sept. 15. All had used milk or cream coming from 1 creamery. The driver of the skim-milk wagon of this creamery had typhoid at a time when it was possible for him to have been the source of the infection. Water infection had been eliminated.	Driver of skim-milk wagon.	Probably in handling milk.	Explosive outbreak limited to users of milk from 1 creamery.	Wm. T. Sedgwick. Mass. State Board of Health Report, 1894, p. 765
133	July, 1894	Torquay . .	36	8	29	Outbreak limited to 1 dairy route almost entirely. Milk was only thing those infected had in common. Farmer's family had typhoid antedating outbreak. 24 families were invaded.	In family of dairyman.	Probably in milking and handling.	Cases followed 1 milk route and were practically limited to it	Dr. Karkeek, M. O. H. Report of M. O. H. Cited by Hart
134	April, 1894. .	Bacup. .	35	5	30	Dairyman was also night-soil scavenger. He supplied 30 families, of which 13 were invaded by typhoid. Sale of this milk was stopped and outbreak ceased	. .	Milkman was also night-soil scavenger	Limitation of outbreak to 1 milk route and cessation of epidemic when dairy was closed	Dr. J. Brown, M. O. H. Annual Report for 1894.

MILK EPIDEMICS Continued.

TABLE I. *Typhoid* Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
135	February, 1894.	Oberschmollen, Gross Ellguth, and Sadewitz.	90			Explosive outbreak of typhoid in every farmhouse but 1 which supplied a certain cheese factory with milk. The households invaded were in the habit of receiving back the whey from milk and using it. The household not invaded did not do this. Those drinking the whey only after it was boiled escaped. Of the first 54 cases reported, 49 lived in houses supplying the cheese factory with milk and taking back whey in return.	....		Explosive outbreak limited to users of certain milk.	Abstract in Pub. Health, Lond. 1894-5, VII, p. 192, of Report by Doctor Reich in Berl. Klin. Wochenschrift 1894
136	October, 1893	University of Virginia.	3	..	3	Cases occurred on a milk route of which the proprietor was nursing his daughter ill with typhoid.	At dairy........	Milkman acted as nurse.	Cases on one milk route.	Dr. J. S. Davis. Univ. of Va. Special Report.
137	August, 1893	Bandon, County Cork.	69	6	60	A milker nursed a typhoid patient. Milk from this farm was sent to a creamery. Of the 24 farms taking their milk to this creamery and receiving back the skim milk 12 were invaded with typhoid. Of the 180 in this same district not dealing with the creamery only 2 were invaded and these cases had eaten at infected houses. Of the 12 farms dealing with	A milker nursed a case of typhoid antedating outbreak. This case was imported.	By milker acting also as nurse.	Limitation of outbreak to certain users of milk.	Dr. J. Welply. Lancet, Lond. 1894. I

						creamery but not invaded some boiled their milk before using and others fed all the skim milk to the stock.				
138	July, 1893 . .	Stockport	9	1	9	All cases among people buying milk at one vendor's.	. . . .	. . . .	Cases limited to users of one milk supply.	Doctor Porter, M. O. H. Public Health, Dec., 1893, and Jan., 1894. Cited by Hart
139	May, 189 . .	Kelso, Scotland. . .	73	8	63	Of the first 18 cases only 1 was not a consumer of the suspected milk. In the first 3 weeks the cases numbered 15, 15, and 22. Of the 48 households supplied by this milk only 3 escaped typhoid. 14 days after closing dairy cases became much less numerous and after a month only 7 cases were reported in the next two months. There had been 2 cases of typhoid at milk farm antedating outbreak. Water used from shallow well polluted by sewage from house.	On dairy farm.	Use of polluted well water.	Outbreak almost explosive. Cases practically limited to users of 1 milk supply. 45 out of 48 families using this milk were affected.	Doctor Oliver, county M. O. H. Annual Report for 1893, M. O. H. Cited by Hart
140	March, 1893. .	Louisville, Ky. .	54		44	Typhoid unduly prevalent in best part of city from a sanitary standpoint. Water and sewage eliminated as causes. Of 54 cases 44 used milk from 1 dairy. This dairy supplied less than one-twentieth of the milk used but had forty four fifty fourths of the cases. Milk was the only thing these cases had in common. Water supply (cistern) of dairy examined and bacillus found by Dr. Wm. Vissman resembling morphologically and otherwise the Eberth bacillus. In one family only 1 person used unboiled milk and she contracted typhoid.	. . . .	Use of polluted water at dairy in washing cans, etc.	Cases practically limited to one milk route. Finding of bacillus, apparently the Eberth bacillus, in dairy water supply.	Dr. Wm. Bailey Kentucky State Medical Society Transactions 1893, n. s. II p. 314
141	1893.	Hoya	150		122	Of 136 households patrons of a certain dairy 49 were invaded. Of 594 other families only 18 were invaded. In houses where the milk was always boiled no cases occurred.	. . . .	. . . .	Majority of cases were patrons of one dairy and where milk was boiled no cases occurred.	Doctor Schlegtendal Vierteljahrsschr. f. offentl. Gesundheitspflege, 1900, XXXII, p. 295

MILK EPIDEMICS Continued

TABLE I. *Typhoid* Continued

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
142	1895	Near Strasburg.			All	All cases had milk from one milkman. 17 per cent of those who drank the milk had typhoid while among 300 others there was no case. Epidemic stopped on withdrawal of milk.			Outbreak limited to consumers of suspected milk. Outbreak ceased when milk was stopped.	Schmidt, Hyg. Rundsch., 94 p. 694. Cited by R. G. Freeman.
143	August, 1892	Dundee	73	11	43	Of 37 cases in August, 19 were among consumers of suspected milk and 24 out of 45 in September. At dairy a shallow well was used for washing cans. After stopping sale of milk and the allowance of a proper incubation period only 8 cases were reported.	Typhoid at dairy.		Outbreak ceased after dairy was closed. Outbreak was explosive and most cases were consumers of suspected milk.	Doctor Anderson, M. O. H. Brit. M. J. 1892, II, 598, 902, 915, and special report M. O. H. Cited by Hart.
144	August, 1892	Torquay	20		15	Explosive outbreak. 6 of the first 7 cases were users of the suspected milk. Son of dairyman had typhoid and wife nursed son and milked cows. Of the 20 cases 15 used this milk and the other 5 may have done so.	On dairy farm	Same person nursing typhoid patient and milking cows.	Typhoid on farm preceding outbreak on milk route. In one house there was only 1 person using this milk, and she was the only one to contract typhoid.	Doctor Karkeek. Report of M. O. H. Cited by Hart.
145	August, 1892	Torquay and St. Mary Church	102	8	102	All cases were among consumers of 1 milk farm where water was used from a polluted well in a confined yard. Cases ceased to be reported 21 days after destruction of well.	....	Probably by use of polluted well water.	—	Doctor Karkeek. Report of M. O. H. Cited by Hart.

146	June, 1892.	Nottingham	7		7	Dairy assistant worked 3 weeks while suffering from fever, vomiting, and diarrhœa. Dairy supplied 26 families.	Probably at dairy.			Boobyer Public Health, 1892, IV, 110. Cited by R. G Freeman.
147	February, 1892.	Oberschmollen, near Oels.	55		52	An explosive outbreak of which all but 3 cases were patrons of one dairy.				Reich. Berl. klin. Wochenschr 1894, S 704. Cited by Doctor Schlegtendal
148	1892.	University of Virginia.	8		8	Outbreak on 1 milk route. Milk cans washed at spring just above which the dejecta from a typhoid patient had been thrown.	Milk farm	Polluted spring.	Cases on 1 milk route.	Dr. J S. Davis, Univ of Va Special Report
149	1892.	Altgalendorf, near Oldenburg.			All.	Numerous cases of typhoid were reported Cases were limited to persons dealing with a certain dairy				Referat in der Zeitschr. f Fleisch. und Milchhyg. 1892, S. 86. Cited by Doctor Schlegtendal.
150	1892.	Aire	36		36	All the cases had used raw milk from a dairy at which the milk cans were regularly washed in a pool of water in which there had also been washed the body clothes of several typhoid cases.		Washing cans in infected water.		Referat in der Zeitschr f Fleisch. und Milchhyg. 1892 3 III S. 22.
151	1892.	Near Leeds			All but 1.	Every case except 1 supplied by milk which had been mixed with contaminated water.				Ballard - Armley Stevenson and Murphy. Hygiene, I, 734. Cited by R G Freeman.
152	1892.	Versmold	34	1	24	Of the people belonging to households which were patrons of the suspected dairy 9.3 per cent fell ill with typhoid; of the nonpatrons only 0.78 per cent were affected.				Doctor Schlegtendal. Vierteljahrsschr. f offentl Gesundheitspflege, 1900, XXXII, p. 295
153	1892.	Croya Isenhagen	11		11	Outbreak limited to patrons of dairy and ceased when proper measures were taken against spread by milk.			Limited to patrons of dairy.	Doctor Schlegtendal. Vierteljahrsschr. f öffentl. Gesundheitspflege 1900, XXXII, p. 295
154	December, 1891.	Clermont-Ferrand	23	6	18	Originated in dairy where proprietor and his daughter had typhoid.	At dairy.			Gayon, etc., Rev. de H., 1892, p 993. Cited by R. G. Freeman.

MILK EPIDEMICS—Continued.

TABLE I. *Typhoid*—Continued.

No.	Date.	Place	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
155	September, 1891.	Edinburgh.	63	. .	63	Child sick with typhoid fever at dairy.	At dairy.	. . . . .	Outbreak limited to patrons of 1 dairy.	Littlejohn, Edinb., Med. Jour. CCXXIX. Cited by R. G. Freeman and Dr G M Kober.
156	February, 1891.	Sutton Coldfield	40	5	40	All cases had contaminated milk. Epidemic stopped by stopping milk supply.	. . . .		Outbreak limited to consumers of suspected milk.	Hill Brit. M. J. 1891, p. 136. Cited by R. G. Freeman.
157	1891.	Affinghausen. . . .	117	. . . .		An epidemic broke out in the neighborhood of a dairy. 47 households were affected, of which 36 were patrons of the dairy, 8 were not, and 3 were doubtful. Of the 76 households in the community 38 were patrons of the dairy, and of these 38 typhoid invaded 22. Of the 38 having no relation with the dairy only 4 were invaded.	. . . .	. . . .	. . . . . .	Becker Generalbericht über das Sanitatswesen im Reg.-Bez. Hannover f. 1889 bis 1891, S. 66. Quoted by Doctor Schlegtendal
158	1891.	Rendsburg. . . .	33	. . .	31	An outbreak occurred limited to patrons of 1 dairy. 31 of the 33 cases were among those having daily relations with the dairy and there were only 2 cases among all the rest of the community.	. . . .	. . . .		Doctor Schlegtendal. Viertel-jahrsschr. f. öffentl. Gesundheitspflege 1900, XXXII, p. 295.
159	1891. . . .	White Church.	. . . . . .		All.	Cases occurred among drinkers of milk from a dairy where a child sick from typhoid was allowed to handle dairy utensils. Milk dealer fined.	At dairy. . . .	. . . .	. . . . . .	Brit. M. J. 1891, II 1179. Cited by R. G. Freeman.
160	February, 1890.	Municipality of	133	9	112	1 dairy supplied 218 families of	On dairy farm. .	By diluting	All the features	Dr J Ashburton

		Randwick, Sydney.				which 67 were invaded by typhoid. Of the 133 cases 112 used this milk. There had been cases of typhoid on this dairy farm preceding the outbreak and water was used from a shallow and polluted well. Other agents as causes of outbreak were eliminated. The above well was closed March 31, and after April 16 no new cases developed on this milk route.		milk with polluted well water.	of the outbreak pointed to the milk as carrier of infection and all other agents as the cause of the epidemic were easily eliminated.	Thompson. Notes and proceedings: Legislative Assembly, New South Wales. Cited by Hart.
161	1890. ..	Langenborn and Bargum (Husum).	48 .			The outbreak continued until attention was drawn to the dairy and proper measures enforced.	. . . .			Doctor Schlegtendal. Vierteljahrsschr. f. öffentli Gesundheits. pflege. 1900, XXXII, p. 295.
162	October, 1889. .	Paterson, N. J. .	34		27	Milk from dairy where utensils were washed and milk diluted with water from well infected by drainage from manure pit, which had been used for the deposit of typhoid fever dejecta. Well closed Oct. 16. No cases developed after Oct 24	At dairy. .	Probably by contaminated water.	Outbreak ceased when well was closed	Dr. J. L. Leal. Trans Med. Soc N. J., 1898, p 176 Cited by Dr. G M. Kober
163	March, 1889. .	Melbourne, Australia	43 .		43	Outbreak explosive, limited to customers of 1 milkman, A, whose son had taken ill with the disease a week before the first cases. Father nursed son and handled and washed cans and milked cows. Epidemic rapidly subsided. 93 families were supplied by this man, A, and typhoid appeared in 33	Son of dairyman living in house with parents	Probably by father nursing son and then milking cows and washing cans.	Explosive outbreak limited to customers of 1 milkman and following case of typhoid in milkman's family	H. B. Allen, M. D. Intercolon. Med Cong. Trans Melbourne, 1889, II, 159
164	September, 1888.	Spalsey, E. S. D. .	12	1	12	All cases developed in customers of 1 milk route. No cases on the routes or among the customers of 9 other milk venders. Milk the only thing in common to all invaded households. The dairy suspected of being cause of outbreak used water from a well located 15 feet from a privy vault. When this well was closed cases ceased to develop. This milkman supplied only 20 families but of these 9 were invaded	. . . . .	Probably by washing cans in polluted well water.	Explosive outbreak limited to 1 milk route, cases having nothing else in common.	Dr. F. J. Walker, M. O. H. Annual Report for 1888. Cited by Hart

MILK EPIDEMICS—Continued.

TABLE 1.—*Typhoid*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
165	July, 1888.	Providence...	96		86	3 cases of typhoid at dairy, 1 a few weeks preceding epidemic.	At dairy.	......	..	Dr. R. G. Freeman. N. Y. Med Rec 1896, XLIX, 433.
166	July, 1887.	Bandon County Cork.	17	2	15	Dairy hand had typhoid. 17 cases developed in town; 15 of these were consumers of milk from this dairy, 1 was brother of dairy hand above mentioned, and 1 revealed no connection. This dairy supplied 30 families, of which 11 were invaded. The cases followed the milk route and were scattered over the town wherever this milk was sold.	At dairy	Probably by milker or in handling milk by infected employee.	Cases followed milk route and were limited to it. Typhoid in dairy hand antedated outbreak on route.	J. J. Welply. Report to local Government Board, Ireland. Cited by Hart.
167	March, 1887.	Minneque.	.	..	All	All cases had milk from 1 dairy. In 1 family all who drank milk raw contracted typhoid, those drinking it cooked escaped. Water supply near privy. Case of typhoid fever previously in family.	At milk farm...	..	Outbreak limited to patrons of 1 dairy.	Dr. G. M. Kober. Sen. Doc. 441, 57th Cong. Cited from R. G. Freeman N. Y. Med Rec 1896, XLIX 433.
168	September, 1884.	Elkton, Md. .	20	.	All but 1.	Sudden outbreak. Typhoid had never before been epidemic in Elkton in the memory of the oldest practitioners. All cases but 1 were supplied with milk from a dairy where there was an undoubted case of typhoid. The 1 exceptional case was imported, having come to Elkton already ill. The cases were widely scattered and had	At milk farm...	Might have been either in milking or by use of polluted water in washing cans.	Sudden and unusual epidemic limited to users of milk from a certain dairy. Outbreak ended when use of milk ceased.	Dr. C. W. Chancellor. 6th Biennial Report Maryland State Board of Health, 1886, p. 92.

						nothing in common but milk. The houses invaded were among the better class of dwellings. There was no public water supply. Milk drinkers alone were attacked. Undoubted case of typhoid at dairy the water supply of which was a shallow polluted well receiving surface drainage. Sale of milk ceased and outbreak ended.				
169	April, 1883	Exeter...	20	3	20	The only cases of typhoid in Exeter were in houses taking this milk which was produced on a farm with a faulty cesspool. After the well was abandoned no more cases developed.	. ..	Probably by use of water from polluted well.	Cases limited to 1 milk route.	Dr. L. Woodman, M. O. H. Brit M. J. 1883, I. 875. Cited by Hart.
170	1883.	Bannockburn. ..		..	.	2 cases of fever in family of dairyman. Most of the persons affected were consumers of this 1 dairy.	On dairy farm		...	Brit. M. J. 1883, I 375. Cited by Hart.
171	1883. ..	Mid - Warwickshire sanitary district	12	1	12	Outbreak limited to consumers of 1 dairy where the farmer died of typhoid and the son also contracted it. The well was contiguous to the cesspool which was leaky and was badly polluted as a result. This water was used to wash the cans. The cases on the farm antedated those on the milk route. The outbreak ceased when the dairy was forbidden to sell milk	On dairy farm.	Probably by washing cans with infected well water	2 cases on farm. The limitation of other and following cases to milk route, and the cessation of outbreak when dairy was closed	G. Wilson, M. O. H. Brit. M. J. 1883, I, 1136 Cited by Hart
172	October, 1882.	Grangemouth .	.	.	All.	Case of enteric fever in house from which milk was supplied and all the cases of enteric fever were found in consumers of this milk	In dairy house .		All cases of fever were in consumers of this milk	Brit. M. J., 1882, II, 911 Cited by Hart
173	September, 1882.	Glasgow				Recent illness of febrile character in farmhouse. 2 suburbs of Glasgow visited by enteric fever, the cause of which was traced to milk from this 1 farm.	On farm producing milk			Dr Russell M. O. H. Brit. M. J. 1882 II, 590 Cited by Hart
174	September, 1882.	Halifax Stone Chair			.	Two suburbs of Glasgow visited by enteric fever; cause traced to milk from infected farm	On farm			Dr Russell M. O. H. Cited by Hart. Brit. M. J., 1882, II, 590

MILK EPIDEMICS—Continued.

TABLE I.—*Typhoid*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
[illegible]	August, 1882	Shelf	11	1	11	Outbreak explosive; all cases occurred between Aug. 24 and Sept. [illegible]. Cases limited to consumers of suspected milk	On farm producing milk there was a case of continued fever in July and another in August. Milk was handled in kitchen of house.	Probably in handling	Presence of fever on farm producing milk. Outbreak explosive and limited to consumers of milk from 1 farm	Dr. Britton, M. D., M. O. H. Brit. M. J. 1882, II, 749. Cited by Hart
176	1882	Glasgow	. . . .	. . . .	All.	Explosive outbreak among consumers of 1 milk supply. No case found other than in consumers of this milk.	. . . .	. . . .	All cases of fever in consumers of this milk	Brit. M. J., 1882, I, 954. Cited by Hart.
177	1882	Halifax Stone Chair	. . . .	. . . .	All.	Sharp outbreak among working class portion of certain district, all being consumers of one milk supply. Infection not traced, but all cases were supplied with suspected milk.			All cases supplied with suspected milk	Brit. M. J., 1882, I, 954. Cited by G. M. Kober.
178	August, 1881	Hawick	. . . .	. . . .		Nearly every case received milk from a farm where typhoid prevailed. Milk supply stopped.	At milk farm . .			Brit. M. J., 1881, II., 273. Cited by R. G. Freeman.
179	1881	Christ Church, New Zealand.	. . . .	. . . .	All.	All houses infected, including a lunatic asylum, were supplied by the same dairy. Dairy premises in a filthy condition.	. . . .	. . . .	Outbreak limited to houses using suspected milk	Brit. M. J., 1881, II, 570. Cited by R. G. Freeman.

TABLE II. *Scarlet fever.*

1	August, 1907	Melrose, Mass	19	14	In August there had been 17 cases reported in different parts of the city. 5 were in 1 family and were probably due to contact. The other 12 were widely scattered and all had the same milkman. 2 suspected cases of scarlet fever were found on the milk farm. Milk from this farm was excluded from sale Aug. 26. In the next 3 days 2 new cases were reported and then the outbreak ceased.	On milk farm	Probably by contact of cases with milk utensils.	Cases practically limited to 1 milk route (the exceptional 5 being in one family). Exclusion of this milk from sale was followed by cessation of epidemic.	Dr. C. P. Holden, Melrose, Mass., Chairman of Board of Health. Special report.
2	June, 1907	Buffalo, N. Y.	17	17	Outbreak on 1 milk route. Origin traced to producing farms on 3 of which cases of scarlet fever were found, or had recently existed	On milk farms		Unusual number of cases on 1 milk route.	Dr. Ernest Wende, Buffalo, N. Y. Special report.
3	May, 1907	Melrose, Mass	9	6	During April and May there had been an occasional case reported and all could be traced to school contact. May 23 2 cases were reported. In the next 2 weeks 7 more cases were reported. Of these 9 cases, 2 developed in houses where there had been other cases, 1 gave a history suggesting contact infection, but the other 6 gave no clew except that they had a common milk supply. Investigation of the dairy showed a milker convalescent and desquamating. Instigation of proper quarantine of milker and disinfection of premises was followed by cessation of outbreak.	Milker on dairy farm.	Milking and handling milk.	Case found on farm. Cases on milk route had not been exposed otherwise and outbreak ceased when this source of infection was eliminated.	Dr. C. P. Holden Melrose, Mass., Chairman of Board of Health Special report.

MILK EPIDEMICS—Continued.

TABLE II. *Scarlet fever*—Continued.

No.	Date	Place	Number of cases	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak	Location of original case or cases causing outbreak.	Manner in which milk was infected	Reasons for believing that milk carried the disease.	Reporter and reference.
4	January, 1907	Boston, Mass.	227		195	Outbreak explosive. 227 cases occurred in 4 days. Of these 195 took milk from "A" while the other 32 cases took milk from 17 different dealers. The water supply was above suspicion. Not all of the milk routes of this dairy developed cases. No case of scarlet fever was found which could be considered the original source of infection. This dairyman also sold milk in Cambridge, Somerville, and Everett, and in the 4 places there were reported during the first 20 days of January, 1907, 717 cases of scarlet fever. In Boston 367 cases, of which 80 per cent were supplied with milk by dairyman A; in Cambridge 152 cases, of which 85 per cent were so supplied; Somerville 155, of which 86 per cent used this milk, and in Everett 43 cases, of which 83 per cent obtained it. Of the 717 cases, 485, or 67 per cent, occurred in the 6 days Jan. 7 to 12, inclusive.	Not known, but thought to be possibly on a farm supplying milk. There were 222 farms supplying this dairy with milk.	Not known.	Its explosive nature and the great number on 1 milk route.	Dr. Carlisle Reed 1906 Report Boston Health Dept., p. 126; also Monthly Bull. State Board Health Mass., March, 1907, vol. 2, No. 3, p. 76.
5	November, 1906.	Pontiac, Mich.	20 (?)	...		Cases originated from dairy where there were several cases of scarlet fever which it had been attempted to con-	At dairy.	By milkers.	...	Dr. Mason W. Gray, Pontiac. Special report.

					ceal. Cases returned to work during stage of desquamation.				
6	February 1906.	Melrose Mass...	20	.	19	Outbreak extended over a period of 3 months, and out of 20 cases in Melrose 19 were among customers of 1 milkman. The outbreak ceased when proper precautions were taken to sterilize bottles and prevent infected bottles leaving houses where the disease existed.	Not found......	Not known, but the evidence would seem to show some spread of the disease by infected milk bottles collected from houses under quarantine	19 cases out of 20 on route of 1 milkman who did not properly sterilize bottles collected from houses with cases, and the fact the outbreak ceased when proper precautions were taken.	Dr. C. P. Holden, Melrose, Mass., Chairman of Board of Health. Special report.
7	October 1905	Clifton	9	....	9	Explosive outbreak limited to drinkers of milk in families all of which obtained milk from the same dairy where there was employed a man 2 of whose children were convalescing from scarlet fever. Precautions were taken, the man removed from the farm, and the outbreak ceased	In family of employee on dairy farm.	. .	Explosive outbreak limited to users of milk from 1 farm, cessation of outbreak when precautions were taken against milk.	Dr. E. M. Smith, M. O. H. Pub. Health. London, 1905-6 XVIII, p. 47.
8	March, 1905	Rochester, N. Y.	27	1	....	Outbreak explosive, cases widely scattered, no social intercourse, other means of contagion eliminated, only thing in common was that most of the 25 families infected took milk produced on a farm where a woman convalescing from scarlet fever returned to milking on Feb. 24, while still desquamating, and that her children had just had scarlet fever. All the above cases were reported between March 1 and 20. This milkman supplied only about 80 families.	In milker on dairy farm	By infected and convalescing milker in milking	... .	Dr. Joseph Roby, Rochester, N. Y. Special report.
9	February 1905.	Mount Vernon, N. Y.	50 (?)	2	45 (?)	About 50 cases of scarlet fever were reported within five days. Of these 45 obtained milk from 1 dairy. The sale of milk by this dairy was stopped and no new cases were reported.	. ..	. ...	Outbreak explosive; all on 1 milk route; epidemic ceased when dairy was closed.	Dr. Arch. T. Banning, Health Officer, Mount Vernon, N. Y. Special report.

MILK EPIDEMICS—Continued.

TABLE II.—*Scarlet fever*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
10	1905.	Cambridge	78			"Milk supply was found to be mainly responsible for the outbreak." Suspected dairy derived part of its milk from a farm where a milker and his child had hands and feet "consistent with the later stage of a scarlatinal attack." This source of milk was shut off and no further cases occurred attributable to milk.	On milk farm . .	In milking probably.	Outbreak pointed to milk and when supply was regulated outbreak ceased.	Dr. Anningson, Pub. Health, London, 1904-5, XVII, p. 795.
11	December, 1904.	Springfield, Mass. .	32	.	31	Of the 32 cases all were reported in a period of 2 weeks. 31 were receiving milk from 1 dairy where was found a young man recovering from a mild case of scarlatina but who had continued at work. The other case seemed to have been contracted from one of the 31.	Milker at dairy .	Milking. .	Outbreak ceased when milk from dairy ceased to be marketed.	Mass. State Board of Health Report, 1904, p. 422.
12	December, 1904. .	Amesbury, Mass. .	43		43	All the patients secured milk from the same farmer.	2 unrecognized cases had existed previously in families supplied by this same farmer, one Nov. 21 and the other Dec. 3.	Either through the infection of empty milk bottles or by the milk distributers themselves.	The preceding facts.	Mass. State Board of Health Report, 1905, p. 579.

13	September, 1904.	Buffalo, N. Y....	14		14	Cases on 1 milk route. Origin traced to farm where cases existed in the family of the farmer and also of the hired man. Milk from this farm was stopped.	On farm... ...	....	Unusual number of cases on 1 milk route.	Dr. Ernest Wende, Buffalo, N. Y. Special report.
14	August, 1904 .	Buffalo, N. Y....	10		10	Explosive outbreak on 1 milk route. It was ascertained that milkman had been delivering milk in bottles to a house containing a case of scarlet fever and removing the empty bottles a few days preceding outbreak. A case also occurred at the dairy which was then closed.	On milk route .	Empty bottles taken from house with scarlet fever.	Cases on 1 milk route. Case at dairy. Outbreak ceased when dairy closed.	Dr. Ernest Wende, Buffalo, N. Y. Special report.
15	February, 1904	Liverpool.. ..	59		59	Explosive outbreak in houses supplied with milk from 1 dairy at which there was a case of convalescing scarlet fever. Proper precautions were taken and the epidemic rapidly subsided. A common milk supply was the only association these cases had to one another.	At dairy	Child convalescent from scarlet fever at dairy.	Explosive outbreak among patrons of 1 dairy. Only thing cases had in common was the milk supply. Outbreak ceased 5 days after measures were taken against the dairy.	Dr. A. A. Mussen, M. O. H. Pub. Health. London, 1903-4, XVI, 687.
16	1904.	Leith.... .	40 +			Explosive outbreak among customers of 1 milk shop, which was connected with living apartments where mother had had and still had a sore throat, of which later developments and slight desquamation indicated a case of mild scarlet fever. Her two children also developed scarlet fever. The outbreak stopped a few days after these cases were separated from the milk shop and proper disinfection performed.	Woman in milk shop.	Handling milk.	Explosive outbreak limited to customers of 1 milk shop where scarlet fever existed. Cessation of outbreak when shop was closed	Dr. Wm. Robertson, M. O. H. Leith. Pub. Health. London, 1904-5. XVII p. 445.
17	April, 1903.. ..	Sunderland.	112 .		.....	Out of 329 families supplied by implicated dairy 70 were affected. There was scarlet fever at the producing farm.	At milk farm....	....	....	Dr. H. Scurfield Special report, 1903. Cited by Swithinbank and Newman.

MILK EPIDEMICS—Continued.

TABLE II.—*Scarlet fever*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
[illegible]	[illegible]	Newton, Mass.	2		[illegible]	Sudden outbreak among consumers of milk from 1 dairy and limited to those drinking the raw milk. It was found that a milker had possibly had a mild attack of scarlet fever.	Milker at dairy had what seemed to have been a mild case of scarlet fever.	Probably in milking.	Outbreak among customers of 1 dairy.	A. W. Russell, Newton, Mass. Special report.
[illegible]	July, 1901.	Beverly, Salem, and Bakers Island, Mass.	63		60	Explosive outbreak. 63 cases reported during first two weeks of July. Cases limited to customers of 1 milkman. This man served milk in Beverly, Salem, and Bakers Island and in each place scarlet fever outbreaks occurred among his customers. It was found that on a farm supplying this milk there had recently been 3 cases of scarlet fever and that 1, a boy of 14 years, had milked while convalescent. Sale of milk stopped July 8. Only 5 new cases developed after July 15.	Milker on dairy farm.	Convalescent doing milking.	Explosive outbreak limited to 1 milk route and following it. Presence of disease on dairy farm, and cessation of outbreak when sale of milk was stopped.	Dr. F. L. Morse, Mass. State Board of Health Report, 1901, p. 564.
[illegible]	[illegible]	East Orange, N. J.	34		32	Outbreak explosive. Within 3 days 16 cases were reported in various parts of the city in 14 families, all of which obtained milk from 1 dairy "B." Milk was the only thing in common to all. During the same 3 days 3 cases were reported in Orange, all obtaining milk from "B" and 15 cases in		......	The cases all being limited to customers of 1 dairy and the disease being present wherever this milk was supplied.	New Jersey State Board of Health Report, 1901, p. 294.

						Newark, 13 of which obtained milk from the same source.				
2	April 1901.	London.	372		284	Explosive outbreak between Apr 25 and May 10. 572 cases reported in involved districts, of which 284 used milk coming from 1 dairy in part or whole. On 1 farm producing this milk cases of scarlet fever were also found. The incidence of the disease followed the routes on which this milk was sold. Milk was stopped and the outbreak ceased.	....	....	Explosive outbreak following distribution of milk from suspected dairy and cessation of epidemic 6 days after stoppage of milk.	Pub. Health London, 1901-2, XIV p. 128
22	February, 1901..	Stroud.......	.	.	13	Explosive outbreak, all cases on 1 milk route and in better part of town. There had been no scarlet fever reported in preceding 3 weeks. The only thing the invaded families had in common was milk. On the farm supplying this milk was found a boy who had been ill with sore throat some days before, and who milked the cows. The boy was removed and in 5 days the outbreak ended.	Probably boy on farm	Probably in milking.	Explosive outbreak limited to customers of 1 dairy, and its cessation when source was removed	J. M. Martin, M. O. H. Pub. Health, London, 1901-2, XIV, p. 158
[illegible]	October, 1900 ..	Clifton. ..	75		66	On suspected dairy farm X, there was at time of outbreak a boy suffering with an illness compatible with mild scarlet fever, and within a week 2 brothers came down with well-marked scarlet fever. Clifton has a population of 47,000. This farm X furnished by 3 routes milk to 209 houses, of which 42 were attacked, furnishing 66 cases. During the same period 85 other distributers supplied 6,922 houses, in which there were 9 cases of scarlet fever.	On dairy farm .	Patient on farm.	Distribution of disease followed that of milk from suspected farm. Outbreak was explosive	Dr. D. S. Davies, M. O. H., Jour. of Hyg. 1901, I p. 388.
[illegible]	[illegible]	[illegible]	7	. . . .	11	Scarlet fever at milk farm; 5 mild cases; when milk supply was stopped outbreak ceased.	At milk farm	. . . .	. . .	Doctor Greenwood, M. O. H. Report 1900. Cited by Swithinbank and Newman.

MILK EPIDEMICS—Continued.

TABLE II.—*Scarlet fever*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
25	April, 1900	Plainfield, N. J.	48	......	46	Outbreak explosive; 41 cases reported in 31 houses, all receiving milk from dairyman W, between April 22 and 29. During this same time 7 cases occurred in North Plainfield, 5 of which got milk from W. April 30 sale of milk by W was stopped and the outbreak ceased.	......	......	Cases limited to consumers of 1 dairy. Epidemic ceased when sale of suspected milk was stopped.	New Jersey State Board of Health Report, 1900, p. 232.
26	April, 1900	Cheadle & Gatley, Cheshire.	52	......	52	Scarlet fever on milk farm. Cases in towns scattered but all had common milk supply	On milk farm.	......	......	Dr. J. H. Godson, M.O.H. Report 1900. Cited by Swithinbank and Newman.
27	February, 1900	Millburn, N. J.	15	......	15	Outbreak explosive; all occurred between Feb. 2 and 6. All took milk from dairyman K. Mr. K supplied 13 families with milk and 8 of these families contracted scarlet fever. No other cases were present in the town.	......	......	8 families infected out of 13 supplied by farmer. No other cases in town; no other explanation.	New Jersey State Board of Health Report, 1900, p. 229.
28	January, 1900	Brighton	25	......	25	Outbreak explosive; cases reported between Jan 10 and 24. Cases all consumers of milk from 1 dairy where cowman had had sore throat and was found desquamating about the hands and feet.	Cowman on farm.	By cowman	Explosive outbreak limited to customers of 1 dairy.	J. M. Martin. Pub. Health, London, 1901-2, XIV, 138.
29	January, 1900	Washington, D. C.	33	......	33	All circumstances pointed to milk as carrier of infection, and it was found that a driver desquamating from scarlet	Driver of milk wagon.	Probably by handling by convalescing driver.	Outbreak ceased after proper precautions were taken against	Report of Health Officer, District of Columbia, 1900, p. 34.

					fever had gone to work at dairy just prior to outbreak.			dairy and the incubation period of those last infected had elapsed.	
30	December, 1899	Edinburgh . . .	42	42	Explosive outbreak; all cases consumers of milk from 1 dairy where a milker was found whose face had peeled and whose throat had previously been sore and swollen. This milker was isolated and in 7 days the outbreak was at an end.	Probably on farm.	Probably in milking.	Explosive outbreak limited to customers of 1 dairy and cessation of outbreak 7 days after isolation of suspected cause.	Sir H. Littlejohn, M. D. San. Rec. Lond., 1900, p. 310, Vol. XXV.
31	August, 1899	Glasgow .	47 . . .	38	Several cases of scarlet fever at milk farm. Mother of sick child also did milking.	On milk farm . .	Nurse milked cows.	. . . .	Report of M. O. H., Glasgow, 1899–1900. Cited by Swithinbank and Newman.
32	March, 1899.	Scotstoun, Renfrewshire.	24 ———	24	Explosive outbreak limited to consumers of milk from 1 dairy farm. Milk from this farm was ordered sterilized before distribution and the outbreak ceased. In 10 days the dairyman discontinued the sterilization and another explosive outbreak of 11 cases occurred among consumers of this milk. Sterilization was resumed and again the outbreak ceased.	Not found	Not known. .	Explosive outbreak limited to users of milk from 1 farm and occurring wherever this milk was sold. Cessation of outbreak when this milk was sterilized. Second outbreak when sterilization of milk was stopped and again its recession when milk was sterilized.	Dr. A. Campbell Munro, M. O. H. Annual report, 1899. Abstr. San. Jour., Glasgow, 1900–1901 VII, p. 442.
33	February, 1899 .	Buffalo, N. Y	20 .	20	Explosive outbreak. 20 cases in 4 days. Source of infection traced to dairy farm where 4 cases had occurred. 1 of them convalescent and engaged in milking and handling cans. Proper precautions were taken and outbreak ceased.	On dairy farm . .	Convalescent milked and handled cans.	Source of infection traced to infected dairy; outbreak ceased when proper precautions were taken.	Dr. E. Wende, Jour. Am. Med. Assn., 1900., XXXIV, p. 151.

MILK EPIDEMICS—Continued

TABLE II.—*Scarlet fever*—Continued.

No.	Date	Place	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
34	December, 1898	Washington, D. C.	65		65	All circumstances pointed to milk as the carrier of infection, and a case of scarlet fever was found in the family of the dairyman.	In family of dairyman on farm	Probably at first from bottles returned from infected houses and later by child of dairyman who was found in desquamating stage on farm.	Outbreak ceased when proper action was taken against dairy farm.	Dr. W. C. Woodward, Health Officer, D. C. Report of Health Officer of District of Columbia, 1899, p 43
35	October, 1896	Norwalk, Conn.	29	....	27	29 cases of scarlet fever were reported between Oct 25 and Nov 9 in 25 families living in 24 houses, cases widely separated in distant parts of town, contact and school infection eliminated; no social intercourse. 27 of the cases obtained milk from milkman H, who supplied only about ⅕ of the milk used in Norwalk. It was found that child of farmer supplying milk to dairyman H had scarlet fever and that other children with the same disease had also been on the farm. Milk from this farm was excluded from sale and epidemic ceased. (The 2 cases not on milk route of H were in 1 family.)	In child on dairy farm and in playmates of this child	Probably accidental from children	Elimination of other causes. Practically all cases on 1 milk route. The finding of case on dairy farm, and the cessation of epidemic when sale of milk was stopped.	Dr. H. E. Smith, Conn. State Board of Health Report, 1897, p 259.
36	May 1897	Montclair, N. J.	20	......	20	The few previous cases which had existed in Montclair were	Unknown	Unknown	The fact that all the cases were	New Jersey State Board of Health

						mostly among those living under unsanitary conditions. In 16 days 20 cases were reported in families living under good sanitary conditions. These families had nothing in common but their milk supply which was the same in all 20. No case of scarlet fever could be found at the dairy. All the cases of the disease were on the same route.			on one milk route which could hardly be a mere coincidence	Report for 1897, p. 111.
37	February, 1895..	Strond-Green.....	200		187	Of 60 cases in hospital 58 had had the suspected milk. On Jan. 8, case of scarlet fever had occurred on the producing farm, followed by sore throat in nurse.	On milk farm			Clothier. Brit. Med. Jour., 1895, I, 549. Cited by R. G. Freeman.
38	February, 1894..	Richmond, Surrey	55		52	Outbreak explosive; all cases reported within 9 days. All but 3 used milk from 1 dairy. There was scarlet fever in vicinity of farms supplying milk to dairy	Probably in vicinity of producing farms.		Outbreak very explosive and 95 per cent of cases were customers of 1 dairy	Doctor Rowland, M. O. H. Annual Report for 1894. Cited by Hart.
39	1894	Lewisham...				Outbreak extensive and sudden; traced to dairy farm on which several cases of scarlet fever existed	On milk farm			Diet. and Hyg. Gaz., 1894, p. 246. Cited by Freeman
40	October, 1893	St. Pancras....	28		28	Cases followed the distribution of milk from a certain farm where 1 of the milkers had scarlet fever	On milk farm	Milker had scarlet fever	Outbreak limited to users of certain milk coming from farm where milkman had scarlet fever	Doctor Sykes, M. O. H. Report 1893. Cited by Swithinbank and Newman.
41	1893	Buffalo, N. Y..	57		57	Explosive outbreak, 57 cases in 26 families all of whom obtained milk from 1 dairy. Investigations revealed 2 cases of convalescents from scarlet fever still in the desquamation stage on the farm producing the milk. One of these was a young man who milked the cows and washed the cans. Proper precautions were taken and the outbreak ceased	Milker on farm	Infected employee in milking and washing cans	Explosive outbreak limited to users of milk from farm where 2 convalescents still desquamating were found and cessation of outbreak when proper precautions were taken	Dr. E. Wende. Jour. A. M. A., 1900, XXXIV, p. 150.

MILK EPIDEMICS—Continued.

TABLE II. *Scarlet fever*—Continued.

No.	Date	Place.	Number of cases	Number of deaths	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference
42	18[illegible]	Vermont	15		15	There were 3 milk routes in town. These cases were among the patrons of 1 route. 1 dairy had in its employ a man desquamating from scarlet fever. 12 of the cases took milk from this man. 1 borrowed this milk from her neighbor, 1 called on a friend and used this milk in strawberries and cream, and the one remaining case associated much with the original case at the dairy and may have been a contact case.	At dairy	Probably in handling milk.	Cases limited to users of a certain milk.	Dr. O. W. Sherwin, Woodstock, Vt. Special report.
43	1893	Vermont	6		6	Milkman while still desquamating milked the cows and delivered the milk. 6 cases occurred on his route.	On milk farm in milker.	Probably in milking		Dr. C. S. Caverly, Pres. State Board of Health, Vermont. Special report.
44	October 1892.	Langham, Essex	5		5	2 families supplied by milkman, both invaded by disease. Cows had an udder eruption.	Suposed to be eruptive udder disease of cows.			J. Cook, M. O. H. Report of M. O. H. Cited by Hart.
45	January, 1889.	Macclesfield	123			Out of 103 families supplied by the implicated milkman 100 were invaded. The diagnoses during the outbreak were scarlet fever 38, diphtheria 2, sore throat 83. Severe sore throat was the prevailing symptom. The attacks were most severe among the great-			Outbreak limited to the users of certain milk.	Doctor Parsons. Loc. Gov. Board Report, 1889, p. 89. Cited by Swithinbank and Newman.

						est users of milk. (This disease was in all probability not scarlet fever.)				
46	December, 1887	Norfolk	5	....	5	Cases among children who had drunk milk from cow with eruption on teats and udder. Cases had an affection of the mouth followed by a vesicular eruption.	Supposed to be eruptive disease of cow.	....	....	Dr. H. Mallins. Lancet, 1888, I, p. 119. Cited by Hart.
47	April, 1887	Stapleton	11	4	11	All the cases were in drinkers of milk from 1 dairy.	....	....	All cases used milk from 1 dairy.	Dr. W. Brown, M. O. H. Monthly Report M. O. H. Cited by Hart.
48	1883	Kingston-upon-Hull.	5	....	5	Child of milkman sick with scarlet fever; 5 cases developed among consumers of milk	At dairy.	....	....	Mason. Brit. Med. Jour., 1883, I, p. 334. Cited by Freeman.
49	1882	Hull	......	....	......	Case of scarlet fever in child at milk shop 3 weeks before being reported. All other foci were traceable to this case	Child at milk shop had the disease.	....	....	J. Mason, M. O. H. Brit Med. Jour., 1883, I, p. 334. Cited by Hart.
50	October, 1881	Greenock	100	....	Nearly all	Nearly all the families attacked took milk from farmer in whose family there had been a recent case of scarlet fever.	In farmer's family.	....	Nearly all cases used milk from 1 farmer in whose family was a case antedating outbreak	Brit. M. J., II, 1881, p. 717. Cited by Hart.
51	April, 1881	Wolverhampton	......	....	All	Milk seller engaged in distributing milk during illness of her children, whom she nursed while ill with scarlet fever. All cases of the outbreak were traceable to her shop.	Children of milk vender.	Possibly and probably by handling of milk by person also acting as nurse.	....	Doctor Love, M. O. H. Brit. Med. Jour., 1881, I, p. 819. Cited by Hart.

TABLE III.—*Diphtheria.*

1	September, 1907.	Warwick, R. I.	67	1	64	Explosive outbreak among consumers of milk from 1 dairy.	On milk farm.	Milker had diphtheria.	Explosive outbreak limited to users of 1 milk supply.	Dr. G. T. Swarts, Providence, R.I. Special report.
2	September 1907.	Pawtucket, R. I.	14	1	11	Outbreak limited largely to consumers of milk from 1 dairy; cases also found on farm supplying milk.	Probably on farm.	....	Outbreak limited largely to consumers of milk from 1 dairy.	Dr. Gardner T. Swarts, Providence, R. I. Special report.

MILK EPIDEMICS—Continued.

TABLE III.—*Diphtheria*—Continued.

No.	Date	Place	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
	April 1907.	Hyde Park, Dorchester, and Milton, Mass.	72		69	Outbreak explosive; all cases occurred between April 12 and 19, and 32 were reported on 1 day, the 13th. 69 of the cases took milk from either milkman A or milkman B, both of whom bought part of their milk from farmer J, whose grandchild took sick with diphtheria Apr. 11. The milk utensils were washed in the house by the same person who nursed the child. The 3 cases not taking this milk were in the same house and secondary to a case reported on April 11 in Milton.	Grandchild of dairyman living on farm.	Nurse of patient washed milk cooler.	Outbreak explosive and found wherever the suspected milk was sold; cases limited to consumers of this milk; case of disease found on farm, outbreak ceased abruptly when milk was no longer sold from this farm.	Mass. State Board of Health Monthly Bulletin for May, 1907, p. 117.
4	October 1906.	Clifton (a suburb of Cincinnati, Ohio).	36	6	36	"Beginning Oct. 3, 1906, within a few days 36 cases of diphtheria developed in the suburb of Clifton within 4 squares of each other. The membrane appeared in all the cases first on the tonsils. There are several reasons for believing the milk was the carrier of the infection. First, each of the 36 cases drank the suspected milk. Second, where only 1 member in a family drank the milk, as happened in several families, only that member contracted	Boy delivering milk from a small dairy in the neighborhood and who was suffering from a "sore throat" but not under the care of a physician.	By being handled by boy with diphtheria.	Without a single exception each of the 36 patients infected drank this milk, and fact that the boy delivering the milk had been sick with a "sore throat."	Dr. L. S. Colter, 3410 Clifton ave., Cincinnati, Ohio. Special report.

diphtheria. Third, in two families who took milk from this dairy but who sterilized the milk before using it, no diphtheria appeared. Fourth, in the case of a small boy who returned home from Europe on the afternoon of Oct. 4, and who on reaching home at once ran over to the cottage of their coachman and there drank a glass of the suspected milk. Diphtheria appeared on the evening of Oct. 6. The following day the coachman, his wife, and his daughter all had diphtheria, all having drank of the same milk. At this same time the writer had 2 cases develop in a boarding house in the heart of the city, several miles distant from the other cases. These 2 persons, mother and child, had daily visited the boy's grandmother in Clifton, who took milk of this milkman and there drank the suspected milk."

MILK EPIDEMICS Continued.

TABLE III.—*Diphtheria*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
5	May, 1906......	Wellsville, N. Y..	84	4	84	"First case was reported May 21," followed by other cases May 23, 26, 27, 28, 29, 30, and June 1, 2, and 4 in all sections of our village. There were 46 cases in 38 families on June 4. Up to this date no case existed in any family whose milk supply was other than the dairy here mentioned. While children in milkman's family were ill the milk was cared for away from the house and the milkers remained away from the house and the sick. After the children were reported well and a faulty disinfection made by the mother, the milkers returned to the house and the milk was again cooled and bottled in the house. 10 days later—that is, 10 days following the first return to the house with the milk—we had our first case of diphtheria. The circumstances and conditions all point to a faulty disinfection as being the cause for the milk's becoming infected.	"In milkman's family 4 children had been ill with the disease in March or April, about 6 weeks or 2 months prior to this outbreak.	"While cooling and bottling and making ready for market or delivery to customers."	"The disease having existed in the milkman's family 6 or 8 weeks prior to outbreak and then the disease occurring in no family excepting those using this milk until we had 46 cases. These all occurred in 14 days. Following these we had other cases due to exposure."	Dr. G. H. Witter, Wellsville, N. Y. Special report.
6	August, 1905....	Rochester, N. Y...	10	1	10	Outbreak explosive; all cases reported between Aug. 16 and	In milker and children on	By milker with the disease.	Outbreak explosive and limited	Dr. Joseph Roby, Rochester, N. Y.

						27, and all on 1 milk route; cases scattered and in 9 different families. Schools were not open; other means of contagion eliminated. Milker on farm producing milk used by these families had clinical and bacteriological diphtheria Other cases also occurred in children on farm.	dairy farm.		to 1 milk route; schools not open; cases scattered; contact eliminated; milk only thing in common; case in milker.	Special report.
7	November, 1904.	Helena, Montana.	19	2	19	Outbreak explosive; all cases reported in 1 week, and all obtained milk from 1 dairy where a child had diphtheria	In child on dairy farm.	Probably from child on dairy farm.	Explosive outbreak limited to milk route of dairyman whose child had the disease.	Dr. T. D. Tuttle, Secretary Montana State Board of Health. Special report.
8	June to October, 1904.	Leith..			All..	An extensive outbreak among families in a good residential district of the city All cases derived milk directly or indirectly from 1 dairy where the cows were found to be suffering with a teat eruption The throats of the dairymen were negative: their hands contained ulcers contracted from the cows The epidemic stopped as soon as proper precautions were taken against this milk Cultures from most of the human cases had shown the positive presence of the Klebs-Loeffler bacillus Cultures taken from the teats showed streptococci and staphylococci and occasionally a bacillus having the microscopical and cultural characteristics of the Klebs-Loeffler bacillus	Alleged to be pustular eruption of cows' teats.	Alleged from diseased teats.	Outbreak limited to users of milk from 1 farm and cessation of outbreak coincidently with precautions against the milk.	Dr. Wm. Robertson, M. O. H., Leith. Pub. Health, London, 1904, XVII, p. 246.
9	June, 1904	Auburn, N. Y....	60	1	45	45 cases occurred on 1 milk route; the other 15 took milk from 13 different dealers. Investigation revealed 2 cases of diphtheria on the milk farm.	At milk farm. .	By a milker. . .	Epidemic ceased when milk was removed from sale and proper precautions taken.	Dr. A. H. Brown, Health officer, Auburn, N. Y. In Annual Report for 1904, p. 17.
10	October, 1903..	Los Angeles, Cal..	35	.....	35	Between Oct. 21 and Nov 1 35 cases were reported from 33 families getting milk from 1 dairy where cultures from the throats of milkers showed Klebs-Loffler bacilli in 3 persons.	On dairy farm, or possibly bottles were infected in houses.	From dairy .	....	Dr. L. M Powers Southern California Practitioner, XIX, 1904, p 113.

MILK EPIDEMICS—Continued.

TABLE III—*Diphtheria*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
11	[illegible]	Cal.	33	..	33	Between Oct. 7 and 17 33 cases reported in 30 families out of 110 families receiving milk from 1 dairy where cultures from throats of family showed Klebs-Löffler bacilli in 2.	On milk farm in milkers.	Possibly original infection came from bottles returned from infected houses.	..	Dr. L. M. Powers. Southern California Practitioner, XIX, 1904, p. 113.
12	[illegible]	Vermont	6	..	6	Son of milkman had a sore throat but continued to deliver milk. Delivered milk from large can, dipping out to each customer the desired amount. Klebs-Löffler bacilli found in boy's throat, cases in houses where he delivered milk, none in houses on same street where he did not leave milk.	Boy who delivered milk.	....	..	Dr. H. D. Holton, Secy. State Board of Health, Vermont. Special report.
13	March, 1901	Montclair, N. J.	15		15	Explosive outbreak, 15 cases of diphtheria reported in 5 days, all consumers of milk from 1 dairy. Examination of employees of dairy showed diphtheria bacilli in the throats of 3. Proper precautions against milk were taken and epidemic ceased.	In handlers of milk at dairy.	In handling milk at dairy.	Explosive outbreak limited to consumers of milk from 1 dairy and the finding of diphtheria bacilli in throats of 3 employees.	New Jersey State Board of Health Report 1901, p. 292.
14	1901 ?	....	5	....	5	2 cows affected with eruptive disease of udder. Then 2 cases of typical diphtheria and 3 of suspicious sore throat appeared among users of the raw milk. Users of boiled milk escaped. Typical Klebs-	From some unknown case to cow's teats.	By milking presumably.	Klebs-Löffler bacillus isolated from milk.	George Dean, M. A., M.B., Charles Todd, M. D., D. P. H. Jour. Hyg. Camb. 1902, II, p. 194.

						Löffler bacilli were isolated from ulcers on cow's teats, from the milk, and from the throat of one of the patients. This ulceration of teats and udders was supposed to be a specific disease, neither diphtheria nor cowpox, and that the diphtheria bacilli constituted a secondary infection.				
15	May, 1900.	Edinburgh.. .	50(?)		50(?)	Explosive outbreak; all cases consumers of milk from 1 dairy farm where the family was found to have several cases of sore throat which bacteriological examination proved to be true diphtheria. Dairy was closed and outbreak ceased.	On dairy farm ..	By infected persons milking and handling milk.	Explosive outbreak limited to users of milk from infected dairy. Outbreak ceased when dairy was closed.	Sir H. Littlejohn. The Sanitary Journal, Glasgow, VII, 1900–1901, p. 211.
16	July, 1899.. ...	Plainfield, N. J..	10	. . .	10	All occurred between July 12 and 20 and received milk from same man. Several cases also occurred in North Plainfield among customers of the same dairy. Investigation revealed a case of diphtheria on the dairy farm in 1 of the farmer's children	Child of dairyman living on farm producing milk.	....	The cases being limited to customers of a rather small milk route, the finding of a case on the farm and the absence of other explanation.	New Jersey State Board of Health Report, 1899, p. 280
17	1899..	Brookline, Mass.	12	. .	12	2 children of milkman had diphtheria and were sent to hospital. Klebs-Löffler bacilli were also found in throats of milkers. In Brookline only 7 or 8 families took this milk and of these 4 developed diphtheria. Cases were also found in Boston on this same man's route. In these 12 cases other sources of infection were not found. All diagnoses were made bacteriologically.	In family of milkman.	Probably by milkers.	Cases followed milk route, diphtheria at dairy.	H. L. Chase. J. Mass. Ass. Boards of Health, Boston, 1900; April, p. 5.
18	December 1898.	Senghenydd....	39	.. . ..	31	Explosive outbreak. Cases widely separated; 31 out of 39 had common milk supply. A bacillus in all respects resembling the Klebs-Löffler bacillus was isolated from the milk.	....	....	Diphtheria bacillus found in milk by Bowhill and verified by Nuttall.	T. W. Thomas, Jour. State Med., London, 1899, VII, p. 705, and Thos. Bowhill, Veterinary Record, London, Apr. 8, 1899, XI, No. 561.

MILK EPIDEMICS Continued.

TABLE III. *Diphtheria* Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases supplied with milk from the same dairy.	Circumstances of outbreak.	Location of original case or cases causing outbreak.	Manner in which milk was infected.	Reasons for believing that milk carried the disease.	Reporter and reference.
[illegible]	May, 1898	Philadelphia, Pa.	15		15	In 1 week 15 cases were reported among consumers of milk from 1 dairy. Houses scattered, children attended different schools, several adults were attacked. Klebs-Loffler bacilli found in culture from throat of child on dairy farm, and also in all cases on milk route.	Probably on dairy farm.	. . . .	Outbreak limited to consumers of milk from 1 dairy, cases synchronous and widely scattered.	Dr. R. L. Pitfield, Univ. Med. Mag. 1897-98, X, p. 669.
[illegible]	August to December, 1892	Limekilns. . . .	28	4	16	At beginning of epidemic most of the cases were in families using milk from a dairy where there was found a case of diphtheria in a boy walking about. A large per cent of the families using this milk were affected.	On dairy farm	. . . .	The early cases found largely among users of this milk, and most families using it were affected.	Doctor Nasmyth, M. O. H. Annual Report for 1892. Cited by Hart.
21	1888	Devonport	61	17	47+	Milk supply of only 50 of the cases could be ascertained, of these 47 were supplied from a common source.	. . . .	. . . .	. . . .	Cited by Swithinbank and Newman. Bact. of milk, p. 349.
22	April, 1883 . . .	Suburb of London.	16	2	16	Explosive outbreak. Cases widely scattered, but all taking milk from 1 dairy.	. . . .		Cases although widely scattered all took milk from 1 dairy.	Doctor Morell Mackenzie. Brit. Med. Jour., 1883, I, p. 874. Cited by Hart.
23	1879.	Kilburn and St Johns Wood.	264	. . . .	. . .	Most of the invaded houses were supplied with milk from 1 farm. Between 13 and 15 per cent of houses so supplied were invaded, whereas only 1.3 per cent of other houses were affected.	. . . .	. . . .	. . . .	W. H. Power. Local Gov't. board report, 1879. Cited by Swithinbank and Newman.

TABLE IV. *Sore throat and pseudo-diphtheria*

1	April, 1905.	Colchester. .	208 .	194	Outbreak explosive; cases confined almost entirely to consumers of milk from dairy M. Cow was found with a mastitis giving milk containing pus and streptococci. This cow was isolated and the outbreak immediately stopped. The cases given are those of which data could be obtained. It was estimated that there had been at least 660 cases in the outbreak. Throats examined showed streptococci. Milk from wagon examined showed pus and streptococci. Milk from diseased cow was filled with pus and streptococci.	Streptococcic mastitis of cow	Diseased udder.	Explosive outbreak practically limited to users of certain milk and coincident mastitis in cow, and cessation of outbreak when cow was isolated.	Dr. W. G. Savage. M. O. H. Pub. Health, Lond., 1905–6, XVIII, p. 1.
2	October, 1904. . .	Paisley.	100+		Epidemic of acute sore throat with severe constitutional symptoms lasting from 2 to 4 days, white diphtheritic-looking membrane formed on throat and pharynx. Bacteriological examinations failed to show the Klebs-Loeffler bacillus, but instead a diplococcus was present. The only thing in common to the cases was the milk supply. An examination of the cattle showed that they had recently suffered from an eruptive disease of the teats, but at the time of examination, which was after the epidemic had practically subsided, the ulcers on the teats had healed.	Alleged to be eruptive disease of cows' teats.	Alleged to be from diseased teats, probably cowpox.	Milk seemed to be the only factor common to all cases.	Dr. A. Robb, M. O. H., Paisley. Pub. Health London, 1904–5, XVII, p. 773.

MILK EPIDEMICS—Continued

TABLE IV—*Sore throat and pseudo-diphtheria*—Continued

No.	Date	Place	Number of cases.	Number of deaths	Number of cases supplied with milk from the same dairy	Circumstances of outbreak	Location of original case or cases causing outbreak	Manner in which milk was infected	Reasons for believing that milk carried the disease	Reporter and reference
[illegible]	May, 1904	Glasgow	[illegible]		[illegible]	Outbreak of sore throat at Belvidere Hospital among the staff, nurses, steward, and patients. Its distribution was such that contact infection was eliminated. The only thing in common was the milk supply. Sterilization of the milk stopped the epidemic. Investigation revealed that herd supplying the milk had an epidemic teat eruption which had appeared a few days before onset of sore throats in the hospital. A similar eruption had broken out on the hands of 4 of the milkers. Other cases of sore throat were found in families supplied with this milk.	Teat eruption of cows	Milking	Outbreak among consumers of milk coincident with teat eruption of cows. outbreak stopped by sterilization of milk.	Dr. A. K. Chalmers, M. O. H. Pub. Health London, 1903-4, XVI, p. 769
4	October, 1903 ..	Woking, Surrey ..	250+		Nearly all.	Explosive outbreak. It was found that the majority of the cases were supplied with milk, directly or indirectly, from 1 farm where 4 cows were found giving milk loaded with pus and streptococci. Sale of milk from this farm was stopped and immediately the outbreak ceased. Farmer had severe sore throat (thought to have been quinsy) about the middle of	On milk farm. .	Either directly from farmer and his family or indirectly by infection of cows' udders.	Explosive outbreak among consumers of 1 milk supply and its cessation as soon as sale of milk was stopped; also finding disease on farm.	R. W. [illegible]. Pierce, M. D., B. S., D. P. H. Jour. of State Med. London, 1904. XII, p. 595.

						September. During October 5 other members of the family had sore throat. Cultures from throats during epidemic showed absence of Klebs-Löffler bacillus and presence of micrococci in groups and chains. 1 strain of streptococci from 1 of the cows was still virulent to mice after 6 weeks.				
5	November, 1894.	Finchley .	..	..	.	Explosive outbreak. Between Nov. 8 and 18 94 per cent of families supplied by this dairy were invaded with sore throat; order issued to boil all milk and outbreak ceased. Cows at dairy were suffering from teat and udder eruptions.	Supposed to be due to teat eruption of cows.	At milking.	Outbreak followed and limited to 1 milk route	Doctor Kenwood, M. O. H. Brit. M. J., 1895, I, pp 1167, 1168 Cited by Hart.
6	August, 1892.	Glasgow ..	224		All.	Outbreak explosive. Between Aug. 6 and 8 between 60 and 70 cases were reported. Milk supply stopped on 8th; cases ceased on 12th. Cows of dairy had a teat eruption from which "Hendon" streptococcus was isolated.	Supposed to be due to teat and udder eruption of cows		Cases limited to drinkers of this milk.	Doctor Russell, M. O. H. Brit. Med. Jour., 1892, II, pp. 432, 666 I, 1893, p 35 Cited by Hart
7	March, 1881.	Rugby .	100		100	Cases limited to users of suspected milk and supposedly caused by using milk of cow suffering with garget.	Supposed to be cow suffering with garget		. Cases limited to users of this milk	G. Wilson, M. O. H. Brit. Med. Jour., 1881, II, p 415. Cited by Hart.

MILK EPIDEMICS—Continued.

[Compiled by S. C. Busey and G. M. Kober.**]

TABLE I.—*Typhoid (Hart[7]).*

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Percent	Circumstances of outbreak.	Reporter and reference.
1	October and November, 1857.	Penrith.					The outbreak, which affected 7 families, was traced to a supply derived from a milkman in whose cottage were cases of typhoid fever. The milk was kept in the kitchen, where the children lay, and the mother, who was the nurse, also milked the cows.	Dr. M. W. Taylor, Edin. Med. Jour., May, 1858; Brit. Med. Jour., Vol. II, 1870, p. 623.
2	July and August, 1870.	Islington, part of .	175	30	175	100	No evidence of typhoid fever at the premises; there was an underground water tank at the milk shop, communicating by means of rat burrows with two old drains, possible overflow of sewage from these into the tank, from which the water was used to wash the milk cans.	Dr. E. Ballard, M. O. H., Brit. Med. Jour., Vol. II, 1870, p. 589; Med. Times and Gazette, Vol. II, 1870, p. 611.
3	July and August, 1872 .	Armley, near Leeds.	107	11			Traced to a milk farm where typhoid occurred in May, dejecta suspected to have been thrown on dung pit; in the latter part of patient's illness copious rains fell and probably washed the germs from the pit or polluted soil into the well, as about this time the cause of the fever began to operate among consumers of the milk.	Dr. E. Ballard, M. O. H., Reports Medical Officer of privy council and local government board, Vol. II, 1874, p. 79.
4	October and November, 1872.	Leeds.	93	14	80	86	Typhoid fever at milk farm since September. Water supply pure, sick room communicated with kitchen and dairy, and the air of these premises common. Kitchen drain communicated with manure heap, and the privy which received typhoid excreta was overflowing.	Dr. M. K. Robinson, M. O. H., Brit. Med. Jour., Vol. I, 1873, p. 68.
5	November and December, 1872.	Moseley and Balsall Heath.	96	10			A case of what was no doubt typhoid fever occurred in a house located between two milk sellers; dejecta thrown into the privy from which the virus must have found access to the water of the milk sellers' wells. One of them polluted the milk, the other made no profession of selling it pure.	Dr. E. Ballard, M. O. H., Report Medical Officer local government board, No. II, 1874, p. 92; Brit. Med. Jour., Vol. I, 1873, p. 68.
6	January, 1873. .	Parkhead, Glasgow. . .	39	6	46	86	Typhoid fever at dairy among the children in December. Milkers also nursed the affected children.	Dr. J. B. Russell, M. O. H., Glasgow Med. Jour.

7	April, 1873.	Chester	15		15	100	Cases of fever at the milk shop in latter part of 1872; a grocery and provision shop used also as a milk house.	Dr. E. Waters.
8	July and August, 1873.	Marylebone and adjoining districts.	244	26	218	89	Occupant of milk farm died June 8 of ambulant typhoid fever; dejecta buried in an ash heap, the soakings from which must have found access to the well used for dairy purposes.	Drs. J. N. Radcliffe and W. H. Power. Report Medical Officer local government board, No. II, 1874, pp. 103–136; Brit. Med. Jour., Vol. II, 1873, pp. 206, 207, 296.
9	July, 1873, to November, 1877.	Ascot.	69		58	84	No case of typhoid occurred at the milk farm till August, 1876, when the epidemic had lasted for three years. Contagion originally reached farm probably through the water entering the well in the yard, carrying the germs from elsewhere, after which the water used for washing milk utensils had an opportunity of specific pollution at the farm.	Dr. E. Ballard. Brit. Med. Jour., Vol. I, 1880, p. 83; Report Medical Officer local government board for 1877, p. 39.
10	August, 1873.	Brighouse, Yorkshire.	68	8	65	95	No typhoid fever at milk farm or dairy; cows healthy, but drank from a cesspool. Complaints of milk smelling badly and becoming offensive after standing awhile.	Dr. T. Britton, M. O. H. Brit. Med. Jour., Vol. II, 1873, pp. 267 and 334.
11	August, 1873.	Wolverhampton.	63	14			Two children of dairyman sick with typhoid fever in August. Well within a few inches of old flat bottomed brick sewer. Epidemic stayed by cutting off the supply of pump water by this milkman.	Dr. J. H. Love, M. O. H. Brit. Med. Jour., Vol. II, 1873, pp. 267 290 334, 417.
12	May and June, 1874.	[illegible] Lane.	65	1	50	76	First person attacked was the dairyman. Wife nursed him and milked the cows. The dairy well upon analysis was found "little better than filtered sewage;" no evidence, however of specific pollution	Dr. R. T. Thorne. Brit. Med. Jour., Vol. II, 1874, p. 391; Sanitary Record, Vol. I, 1874, p. 214.
13	June, 1874.	Taunton.	5		5	100	No evidence of typhoid fever at the source of milk supply. Well water of the dairy subject to 'fearful contamination with sewage.'	Dr. H. J. Alford, M. O. H.
14	August, 1874	Queensbury	36	1	34	94	Farmer's wife sickened of typhoid during outbreak. Well close to house; drain roughly made of stone. Earth between drain and well saturated with sewage.	Annual Report of Medical Officer of Health for 1874.
15	October and November, 1874.	Dundee	19	4	19	100	Typhoid fever cases at farm, 4 patients occupied a bedroom adjoining the milk store. Well water reported to be contaminated with the products of decomposing organic matter of the nature of sewage.	Dr. G. C. Pirie, M. O. H. Brit. Med. Jour., Vol. I, 1875, p. 225.
16	February and March 1875.	Crosshill, Renfrewshire.	153				Two of the farm children had suffered from the disease; dejecta thrown either on the manure heap or into the ditch. Nurses also connected with the collection and disposal of the milk. Well water quite impure.	Drs. H. D. Littlejohn and E. Duncan. Brit. Med. Jour., Vol. I, 1875, p. 391; Sanitary Record, Vol. II, 1875, p. 61.

Trans. Internat. Med. Congress. London, 1881, IV, p. 491.

** Report of health officer of District of Columbia 1895.

MILK EPIDEMICS—Continued

TABLE I. *Typhoid* (*Hart*)—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Percent.	Circumstances of outbreak.	Reporter and reference.
17	August, 1875	Jarrow	34	2	31	91	Six of the farmer's family, including himself, found ill with typhoid. Direct communication between dairy and sick room. Dairy also used as a wash house. The daughter acted as nurse and milkmaid.	Dr. John Spear, M. O. H. Brit. Med. Jour., Vol. II, 1875, p. 372; Sanitary Record, Vol. III, 1875, p. 195.
18	September, 1875	Glasgow	59		58	98	Two cases of typhoid at farm. Washing for patients done on August 3, 10, and 27, in a wash house, closely situated near the pump well. Water quite impure.	Dr. J. B. Russell, M. O. H. Brit. Med. Jour., Vol. II, 1875, p. 535.
19	September, 1875. .	Glasgow . .	121	[illegible]	98	81	Traced to the same milk supply as the epidemic No. 18.	Do.
20	January and February, 1876.	*a* Eagley and Bolton	105	13	.		No typhoid fever cases at farm, which, however, depended for its water supply upon a brook which had been fouled with the excrement of men engaged in building a mill 200 yards off. There was "evidence that some individual who had used the stream had suffered from diarrhea."	Dr. W. H. Power to local government board; J. Robinson, M. O. H. Brit. Med. Jour., 1876, pp. 201, 233, 273, 293, 491.
		b Bolton . .	144	8	. .		(See above.)	
20*a*	February, 1876.	Greenock	20	.	.		A farmer allowed a case to be brought into his house, and after a while 3 servants and several members of his family were taken sick with enteric fever, and communicated the disease to over 20 consumers of the milk.	Brit. Med. Jour., Vol. I, 1876, p. 425; Sanitary Record, Vol. IV, p. 234.
21	Autumn, 1876.	Churwell and Morley. .		9	.		A case of fever at the farm, well water unfit for drinking purposes, but farmer denied having used it for dairy purposes.	Dr. J. C. Clarke, M. O. H.
22	November, 1876.	Great Coggeshall .	28	. . . .	28	100	Imported case of typhoid fever at dairy, dejecta thrown in a drain, emptying into a brook which was used for dairy purposes.	Dr. R. T. Thorne. Official report.
23	December, 1876.	Salford .	13	. . . .	13	100	Sixteen cases of typhoid at the farm within twenty years. Well close to privy cesspool, and a yard or so off was a sink for dirty water.	Dr. J. Tatham, M. O. H. Annual Report of Medical Officer of Health for Salford, 1875-76.
24	December and January, 1876–77.	Barrowford (Lancashire)	57	7	57	100	Recent cases of typhoid at farm. Milk tins washed with the same dishcloth as used among the fever patients; farmer nursed children and milked cows.	Dr. T. Dean, M. O. H. Medical Times and Gazette, Vol. I, 1877, p. 72.

25	1877	The Gurnos Ystalyfera	7	.	7	100	Milk dealer's son sick with typhoid fever. Milk stored in a pantry leading out of the living room of a small, overcrowded house.	Dr H F. Parsons. Report on sanitary condition of Pontardawe rural sanitary district, 1880.
26	January, 1877.	Greenock.	20	2	16	80	No details....	Dr J. Wallace, M. O. H. Brit. Med. Jour., Vol. I, p. 118.
27	February, 1877..	St. Pancras part of northeast district of parish	35	2	30	85	Sudden and explosive outbreak traced to a milk supply, with no evidence of enteric fever at milk shops or farms. Water supply contaminated with filth.	Dr. T. Stevenson, M. O. H. Brit Med. Jour., Vol. I, 1877, pp. 275 and 329.
28	August, 1877	Edinburgh, Coltbridge		(5)			A case of typhoid fever at dairy communicated the disease to over twenty families.	Brit. Med. Jour., Vol. II, etc., 1877, p. 392.
29	October and November, 1877	Tunbridge Wells	68				Milk supplied from various sources, no typhoid fever at the farms, but at one of them the sewage of the town flowed through the cow yard; in the village there had been cases of typhoid fever.	Dr. W. H. Rix, M. O. H
30	December and January, 1877-78.	Glasgow and Hillhead.	166	16			Typhoid fever at one of the supplying milk farms; nursing performed also by dairy hands, dejecta thrown into a channel running on each side of the central passage provided in byres for cattle droppings. From the middle of the byre the washing house was entered, and through this the milk house.	Dr. J. B Russell, M. O. H. Brit. Med. Jour., Vol I, 1878, pp. 101, 165, 270
31	January to March, 1878	Morsside, near Manchester	32	3	29	90	Two deaths from typhoid at farm in February; well in close contiguity to ash pits, and water found to be sewage polluted.	Dr. E. Sutcliffe, M. O. H. Med Times and Gazette, Vol. I, 1878, p. 517
32	July and August, 1878	Bristol⁶	131	12	131	100	A young lady visited the farm in June, just convalescing of typhoid fever. One of the farm servants ill August 1. Cesspool overflowing and its contents were traced by a recurrent course to well, which was used for dairy purposes.	Dr. D. Davies, M. O H. Brit Med Jour., Vol II, 1878, p. 226. Sanitary Record, Vol. II, 1878, pp 160-166.
33	August, 1878.	Croydon	48		37	77	A sudden and explosive outbreak traced to a milk supply, but no evidence of enteric fever at the source of supply	Dr. C. W. Philpot, M. O. H. Annual report for 1878. Brit. Med. Jour., Vol. II, 1879, p. 675.
34	September, 1878..	Portsmouth	153		78	64	Farmer's children had typhoid fever and no doubt poisoned the well, for two children who were out walking and drank water from this well were subsequently attacked. Milk supplemented from another farm, where well was within a few feet of cesspool of a common privy.	Dr. G. Turner. Brit. Med. Jour Vol. II, 1879, p. 675
35	September, 1878.	[illegible] near Glasgow	40	.	40	100	Infected clothing brought to dairy farm to be washed. On the fourteenth and fifteenth days later symptoms of enteric fever appeared in persons receiving milk from this farm; also a convalescing child brought to farm. Water supply on premises deficient; shallow dip well but not used for drinking purposes.	Dr J. Christie Sanitary Record, Vol. IV, p. 342.

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[1] Washington street epidemic.
[2] And 30 suspicious cases.
[3] Pollock Shaw's road and Kingston epidemics.
[4] A great number.
[5] Several deaths.
[6] There was also a supposed outbreak of milk typhoid at Bristol, 8 cases in 5 houses in spring of 1880. Their common milk supply was the only connection.

MILK EPIDEMICS—Continued.

TABLE I. *Typhoid* (*Hart*)—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Per cent.	Circumstances of outbreak.	Reporter and reference.
36	October, 1878	Perth	—		......	.....	All the families in which the disease appeared had their milk from one dairy.	Brit. Med. Jour., Vol. II, 1878, p. 645.
37	December (Christmas), 1878	Dublin	.		67	100	A probable case of typhoid at dairy in November and middle of December. "A strong wind blowing into the yard would certainly waft particles of coal ash, etc., from the dung heap. * * * to these minute portions of human excreta might have adhered." Nurses also connected with dairy.	Dr. C. A. Cameron, M. O. H. Dublin Jour. of Med. Sci. July, 1879, Pt. I.
38	November, 1878	Huncoat	12		12	100	Children of farmer sick with typhoid fever. Father would nurse the children and also attend to the cattle.	Dr. T. Dean, M. O. H. Sanitary Records, Vol. IV, p. 362.
39	February, 1879	Chichester	50	[illegible] ...	......	......	Milking hovel near a stream which receives large quantities of filth. Milkman washed udders of cows with water from this stream, which probably at the time contained the specific poison. Privy pit only 8 yards from well, but no history of any recent typhoid at the farm.	Dr. Hubert Airy. Brit. Med. Jour., Vol. II, 1879, p. 475
40	October, 1879	Bristol	—	......			Milk traced to a suspected farm where there was no enteric fever, but water from pump in the dairy absolutely stunk when pumped, and was described as "simply poisonous."	Dr. Davies, M. O. H. Brit. Med. Jour., Vol. II, 1879, p. 625.
41	January, 1880	Penzance	26	4	26	100	Three cases of typhoid at the farm. The same person who milked the cows and attended to washing of dairy utensils also nursed the patients.	Dr. G. B. Millett, M. O. H. Brit. Med. Jour., Vol. II, 1880, p. 37.
42	April, 1880	Glasgow	508	69	373	73	Dairyman of the farm sickened with enteric fever in March. Subsequently some of the children took sick and lay in bedroom next the kitchen; also dairymaid was taken sick and occupied a room above milk and wash house. Soiled discharges from sick bed washed at dip well, probably also used for other domestic purposes.	Dr. J. B. Russell, M. O. H. Brit. Med. Jour., Vol. I, 1880, p. 985.
43	April, 1880	Possilpark, Glasgow	92	....	90	97	(See above, No. 42.) Dairyman supplied milk shops in Possilpark also.	Dr. J. Christie. Brit. Med. Jour., Vol. I, 1880, p. 864.
44	July to September, 1880	Millbrook, Cornwall	19	......	19	100	Six cases of typhoid fever within three weeks at milk seller's house; milk kept in a filthy apartment near a badly trapped and very	Dr. E. Ballard. Brit. Med. Jour., Vol. I, 1881, p. 20.

							offensive drain inlet, which drain communicated with another which had received infected exereta.	
45	September, 1880....	Rochdale............	35	9	26	74	In a cottage between the farmhouse and shippen a woman had been suffering from typhoid fever; her excreta were thrown over the wall opposite the door into a cess pool, from which the dip of the soil inclines toward farm well. Cattle also waded about this cesspool. Milk probably diluted.	Dr. Joseph Henry, M. O. H. Brit. Med. Jour., Vol. II, 1880, p. 597.
46	September and October, 1880.	Portsmouth, Cambridge Barracks.	7		7	100	Milkman's son had fever with typhoid symptoms in a room upstairs, and dejecta had to be carried through the back kitchen, used also as dairy. Infection either caused by absorption or by the act of milking being performed by attendants on the sick boy.	Surg. Maj. Jameson. Brit. Med. Jour., Vol. I, 1881, p. 61.
47	October, 1880..	Bridlington. . . .	48	8	48	100	Convalescent from typhoid fever visited the dairyman's house, probably in September. The outbreak occurred in the early part of October. Dairy well close to manure pile; privies only 13 yards distant; water evidently largely contaminated by sewage.	Dr. I. Allison, M. O. H. Brit. Med. Jour., Vol. II, 1880, p. 786.
48	October, 1880..	Marylebone (Clifton Hill, etc.).	9	9[1]	9[1]	100	Nil. All cases occurred about the same date. Invaded houses not on the same side of street, nor adjacent. Five out of six households invaded had their milk from one dealer.	Dr. A. W. Blyth, M. O. H. Brit. Med. Jour., Vol. I, 1881, p. 61.
49	October, 1880 .	Southport.	32	2	32	100	No typhoid fever at dairy, but well in close proximity and exposed to excremental pollution; water declared to be nothing but liquid sewage.	Dr. H. H. Vernon, M. O. H. Brit. Med. Jour., Vol. II, 1880, pp. 820-934.
50	October and November, 1880	Worthing....	44	8	44	100	A case of enteric fever in the house; excreta thrown into a defective drain, which was near the well used for dairy purposes; distinct evidence of soakage from this drain into well.	Dr. C. Kelly, M. O. H. Brit. Med. Jour., Vol. II, 1880, p. 934.

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[1] Several cases.

MILK EPIDEMICS—Continued

TABLE I.—*Typhoid (Busey and Kober).*

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Per cent.	Circumstances of outbreak.	Reporter and reference.
51	1868	Dover					Investigation convinced reporter that the milk was the vehicle of the poison and that it became infected by absorption and not through contaminated water.	Dr. M. K. Robinson. Trans. Int. Congress for Hygiene and Demogr., 7th sess., 1891, sec. 3, p. 170.
52	August 28 to September 3, 1872.	Bergen	18		All	100	Enteric fever at farm. Wife acted as nurse and distributed the milk. Explosive outbreak. All cases taken sick between August 28 and September 3.	Dr. Holmboe. Nork Mag. f. Laegerk., 1873, p. 654. Hirsch Handbuch, Vol. I, p. 683.
53	Summer, 1875.	[illegible]	2		All	100	Enteric fever at milk farm. Well highly polluted with refuse. Water used for cleaning milk utensils. No new cases after September 5, when customers stopped purchasing milk except in one family, who continued to buy the milk.	Dr. Lübe. Allgem. Zeitschrift f. Epidem., 1876, Vol. II, p. 298.
54	November, 1878.	Aberdeenshire	2				Piggery close to milk room; bad drain passed under the pump, whence the water for household was procured.	F. A. McEwen. London Practit., 1881, XXVI, 161-164.
55	January, 1879.		15				Utensils washed from a well close to and under the level of the dunghill, and open to any sewage that might percolate in that direction.	Do.
56	January, 1881.		-				Refers to several instances in which "milk had been liable to contamination either directly through persons suffering from the disease or indirectly through sewer emanations or water charged with the specific infective element of the disease for which the milk may also have acted as a cultivation fluid."	Dr. W. N. Thursfield. Sanitary Record, London, 1880-81, n. s., II, 243.
57	March, 1882.	Leicester Infirmary	12	2	12	100	A fatal case of typhoid fever at dairy. Polluted well. All patients had used unboiled milk.	W. Elgar Buck. Mid. Med. Misc. Leicester, 1883, II, p. 73.
58	May and June, 1882.	Glasgow	60	6	50	85	Nearly all cases occurred within one week in May, none since June 1, "and its area has been most distinctly marked out in relation to the milk supply."	Dr. J. B. Russell, M. O. H. Brit. Med. Jour., Vol. II, 1882, July 8.
59	June, 1882	Allegheny City, Pa.	40	4		...	Typhoid fever at dairy. Well only 50 feet from privy vault; the latter was full and higher up on the hill than the well.	Dr. D. N. Rankin. Pittsburg Med. Jour., 1883, III, 289-292.

60	June, 1882.	Clapham.	20	.....	19	95	All cases taken sick within twenty-four hours, all supplied with one exception, with milk from same dairy, health officer unable to explain milk infection.	Brit. Med. Jour., 1882, Vol. II, p. 216.
61	July, 1882.	Halifax Stone Chair..	11	1	11	100	Two cases of probable typhoid at farm. Father of farmer's wife arrived July 11, taken sick July 21, no medical attendant. His wife came to nurse him August 14, taken sick August 21; died September 6. Unsanitary condition at farm, untrapped drain in room where milk was stored; polluted water.	Dr. Britton. Brit. Med. Jour., 1882, Vol. II, p. 749.
62	November, 1882	Newton Heath.	60	....	...	.	Of the first 16 cases 12 consumed milk from the same dairy, 2 obtained their milk from shops, and 2 from still other sources, no details, doubtful connection.	Henry Tomkins and James Niven. London Lancet, 1883, Vol. I, pp. 360, 611.
63	July, 1883.	Goteborg. ...	4	..	4	100	Typhoid cases at milk farm and unsanitary conditions.	Dr. E. Almquist. Vrtljschr. f. Gesundhtspfl., 1889, XXI, 327.
64	January, 1883	Cologne.... .	270	.	.....	..	The cases were distributed in 54 households, all situated in the best part of the city. Typhoid among servants at milk farm, polluted water used in cleaning utensils.	Dr. B. Auerbach. Deutsche med. Wochenschrift, Berlin, 1884, X, 709.
65	February, 1883	Gateshead .	44	6	44	100	All in 30 households supplied with milk from a farm where enteric fever prevailed among the children; the mother nursed and also assisted in milking and dairy work. Utensils kept in a dirty scullery.	Charles Green. London Lancet, 1883, Vol. II, 986.
66	July to October, 1883...	St. Pancras. .	431	62	....	..	Epidemic invaded 276 families, all using milk from a particular dairy farm where enteric fever started in a boy who arrived July 6, and sickened July 16.	Shirley F. Murphy. London Lancet, Vol. II, p. 652.
67	October, 1883	Dundee.	102	.. ..	..		Disseminated by the sale of milk from a dairy kept by a man of whose family several members were sick with typhoid fever.	Brit. Med. Jour., 1883, Vol. II, p. 839.
68	October and November, 1883	Englewood, N. J. ...	10	... ...	10	100	Typhoid fever case at dairy, a woman who assisted in nursing also helped to wash milk utensils.	D. A. Baldwin. Med. Record N. Y., 1883, XXIV, p. 585.
69	October and November, 1883	Port Jervis, N. Y. .	150	17	.....		Three cases of typhoid fever at milk farm in August and September. The bulk of epidemic cases occurred between October 24 and November 15, the sale of milk having been stopped November 4.	Dr. A. P. MacDonald. N. Y. Med. Times, 1883-84, XI, p. 328.
70	December, 1883. ...	Aberdeen	25	2	25	100	Daughter of the owner of the milk farm reported to have been ill with diarrhea, water from an open ditch polluted with sewage and the dejecta of a previous case of typhoid fever, located above the farm, had been used for dairy purposes. Milk supply diluted.	Dr. Simpson, M. O. H. London Lancet, Vol. I, 1884, p. 487.
71	January and February, 1884	Upsala, Lakare Foren	42	.	42	100	Typhoid at milk farm and bad, unsanitary conditions.	Ernst Almquist. Vrtljschr. f. Gesundhtspfl., 1889, XXI, 327.

¹ Families.

² Several cases.

MILK EPIDEMICS—Continued.

TABLE I.—*Typhoid (Busey and Kober)*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Per cent.	Circumstances of outbreak.	Reporter and reference.
72	May [illegible] 1884	St. Albans	131	23			Of 396 houses supplied with the suspected milk, 86, or 21.7 per cent., were infected. Some of the milk sent to London affected consumers there. Milk obtained from a farm where cases of typhoid had occurred	Shirley F. Murphy. Report Medical officer local government board, 1884, Brit. Med. Jour., 1884, Vol. I, 1162, Vol. II, p. 1086.
73	October, 1884	Tweedmouth	23		23	100	All due to milk sold where there was typhoid fever at cow keeper's house, for which he was fined 3 guineas.	Sanitary Record, London, n. s., 1884–85, p. 204.
74	August and September, 1884	Belvidere, Royal and Western infirmaries, Glasgow	145	32			Traced to a particular milk farm where dairymaid took sick at the same time as the outbreak in Glasgow. Prior to this cattle had suffered from a febrile disease attributed to drinking sewage water, enteric fever endemic in adjacent villages; difficult to say whether infection originated with the cows or had been conveyed from another focus.	Dr. J. B. Russell, M. O. H. Brit. Med. Jour., 1884, II, 626, 724; Sanitary Journal, Glasgow, 1884–85, n. s., VIII, pp. 225–239.
75	October, 1884	Derby	40		40	100	Sudden outbreak among customers of a particular dairy, where 4 cases of typhoid fever had previously occurred. Well liable to gross pollution, being situated on the brink of a ditch which received the drainage from the farmhouse.	Brit. Med. Jour., 1884, Vol. II, p. 786.
76	November, 1884, to March, 1885	Groningen	58		46	79	Infected well water at dairy	Dr. Ali-Cohen. Nederl. Tijdschr. v. Geneesk., Amster., 1887, XXIII, 2d, pp. 78, 84.
77	December, 1884	Aberdeen	65	7	43	66	Numerous sources of contamination at the milk farm; well polluted	Dr. Simpson. Brit. Med. Jour., Vol. I, 1885, p. 193.
78	February, 1886	Leichhardt, Australia	38	5			Sewage polluted well at dairy	J. Ashburton Thompson. Austr. Med. Gazette, Sidney. 1885–86, Vol. V, p. 265.
79	July, 1886	Swanage, Dorset					This epidemic of typhoid fever at its commencement was associated with the use of milk from a dairy situated near a polluted brook, and no other water supply was on the premises.	Mr. W. Harvey. Report medical officer local government board, 1886, No. 16, p. 294.
80	July, 1886	Lancing College, Shoreham	14 80 to 100	2	14	100	Outbreak originally traced to cream derived from a certain dairy, where no other evidence could be found than a liability of the well to pollution.	Dr. C. Kelly. London Practit. 1886, XXXVII, pp. 223–231.
81	October, 1886	Carlisle	30		24	79	Traced to a dairy where typhoid cases had existed, preceded by a febrile disorder among	William Brown. Sanitary Record London 1887–88, n. s., IX, pp.

								the cows; water supply and sanitation being quite good.	10-15; Practit., London, 1888 XV, pp. 383 392.
82	November and December, 1886.	Cambridge, Mass. .	73		..			The epidemic invaded 36 families, and was traced to a certain milk farm, where a child was ill with typhoid fever; the father had entire charge of the nursing, emptied the exereta, and also prepared the milk for the market.	Charles Harrington. Boston Med. and Surg. Jour., 1888, CXIX, pp. 49-52.
83	February 1887 ..	Göteborg . . .	43		.	43	100	Affected 34 families, all supplied with a particular milk. Typhoid fever at milk farm, and suspicious sanitary conditions.	Dr. Ernst Almquist. Vrtljschr. f. Gesundhtspflg., 1889, XXI, pp. 327 338.
84	August 1887	Goteborg .. .	5	. .		5	100	In 3 families; typhoid fever at farm. . . .	
85	1887	Denmark. . .	.		. .		. .	Dr. Lehmann, of Copenhagen, before the International Congress of Hygiene and Demographie held at Wien, 1887, described 2 epidemics of typhoid fever traced to a certain creamery, and pointed out the difficulty of tracing infection when milk is received from a number of farms and mixed.	Deutsche med. Wochenschrft., 1889, vol. 15, p. 17.
86	March, 1888	Goteborg.	4		. . . . .			Typhoid at milk farm, and bad, insanitary surroundings.	Dr. Ernst Almquist Vrtljschr. f. Gesundhtspflg., 1889, XXI, pp 327 338.
87	1888	Washington Heights, N. Y.			. . .	.		Dr. Edson is quoted as having reported this epidemic of a disease resembling typhoid fever confined to the customers of a certain milkman. On careful inspection of the cows 1 of them was found to be suffering from a loathsome abscess of the udder. The cow was being milked into the common pail. No other cause could be found, and the sickness speedily stopped when this cow was quarantined	Brooklyn Med. Jour., 1888, Vol. 1 p. 182.
88	July to December 1888.	Spennymoor, Durham	25	5	.			Outbreak occurred in 19 families, 11 of which were supplied with milk from a dairy where typhoid fever and evidence of polluted water were found	Dr. David Page. Public Health, June, 1889; Lancet, London 1888, Vol. II, p. 941.
89	1888	Evesham ..	6	1		5	83	Typhoid at dairy, milk adulterated with polluted water.	Dr. Fosbroke. Public Health, February, 1889.
90	1889	Country town in New York	200 nearly					Investigation showed that only the customers of a certain milkman were affected. His well was contaminated by the drain of a neighbor's house in which typhoid had recently occurred. Water used to wash milk cans, and possibly also for adulteration.	Dr. Wm. M. Smith, quoted by Dr Cyrus Edson. Med. Record N. Y., XXXV, 1889, p. 10
91	1889.	St. George, Hanover Parish				.		Dr. Barry, medical inspector, reports to the local government board on this sudden and localized outbreak of enteric fever, which he attributed to temporary admixture of infected milk with the usual supply, and also refers to nuisance from sewer ventilators, etc.	Report Medical Officer local government board, 1889, p. 47.

MILK EPIDEMICS Continued

TABLE I. *Typhoid Busey and Kober* Continued

No.	Date	Place	Number of cases	Number of deaths	Number of cases among milk consumers	Per cent	Circumstances of outbreak	Reporter and reference
92	February to April, 1889	Dundee	23		23	100	All cases occurred among the customers of a particular dairy, and the most searching inquiries failed to find any trace of disease among the persons handling the milk or in the household, but 1 of the milch cows was suffering from a peculiar teat eruption and as the disease declined upon stoppage of the milk, April 15, Dr. Anderson feels justified to regard the cow as an etiological factor. Other sanitary improvements were made in connection with sewer	A. M. Anderson, Brit. Med. Jour., London, 1889, II, p. 467.
93	February, 1889	Stirling	40	4	40	100	Typhoid fever at milk farm, polluted water, air of the milk house liable to contamination. The epidemic affected especially families supplied with milk which had been kept overnight in the milk house	Dr. McFadyan. Brit. Med. Jour., London, 1889, Vol. I, p. 1250
94	March, 1889	Strand district, London	10		10	100	Dr. Conway Evans, the medical officer of that district, reports that he has traced 10 cases of typhoid fever to the milk supply, and was ordered to visit the farm and take necessary steps	Brit. Med. Jour., 1889, Vol. I, p. 725.
95	[illegible], 1889	Svarteborg, Sweden	104	11			Typhoid fever cases at milk farm; contaminated water used for dairy purposes, also for adulteration of milk.	Ernst Almquist. Zeitshft fur Hygiene, Leipzig, 1890, Vol. VIII, 137-140
96	July, 1889	Belgard	11		11	100	All the 11 typhoid fever cases had obtained their milk from a poor woman, the owner of a single milch cow, whose child was ill with typhoid fever, the milk being kept in a safe in the sick room, it being the only room at their disposal.	E. Roth. Deutsche Vrtljschr. f. offentl. Gesundhtspfl., 1890, XXII, pp. 238-245
97	June, 1889	Leeds	120	2			No details as to the condition of dairy farms given. Cases occurred in the best residential part and were traced to a particular milk supply.	Dr. Goldie, M. O. H. Brit. Med. Jour., 1889, Vol. II, p. 110.
98	November, 1889	York	120				Three cases of typhoid fever had occurred at the milk farm. Inspection revealed a probably infected well close to the privy, milk vessels kept close to privy and milk adulterated with 10 per cent of polluted water.	S. W. North, M. O. H. The Practitioner, London, 1889, XLIII, 393-400

99	January to May, 1890.	Genève	63				The epidemic was traced to a particular dairy, where the most unsanitary conditions were found. Men were seen spitting on their hands while polishing milk cans. There was also evidence of reckless watering of the milk with polluted water.	Dr. Vincet. Epidémie typh. propagée par le lait, Genève, 1890, p. 15.
100	May, 1890.	Forfar	36				Three cases of typhoid fever at the dairy from whence milk was supplied to 28 families. Milk exposed to the contamination of an infected drain.	Dr. Murray, M. O. H. Sanitary Journal, Glasgow, 1890-91, n. s. XIV, p. 113.
101	May, 1890.	Nottingham	7		7	100	Nephew of milkman sick with walking typhoid fever; continued at work. Milk supply stopped June 20; after June 26 no fresh cases occurred.	Dr. Philip Boobyer, M. O. H. Annual Report for 1890, Public Health London, 1891-92, IV., p. 110
102	June, 1890	Waterbury, Conn	50		41	82	Typhoid-fever cases at the milk farm from which at least 41 cases had consumed milk. One of the farm hands continued to work in the care of cans and at milking for a week before giving up; he also defecated in the cow stables, throwing the stools into the barnyard and thus infecting material everywhere.	Dr. Herbert E. Smith. Sanitarian, N. Y., 1890, XXV, p. 298-308.
103	July, 1890.	Sittensen, Hannover.	103				This epidemic affected only persons who had drunk water from a specifically infected well, or skimmed milk from a certain creamery supplied by 70 to 80 milk producers, and the evidence appears to indicate that this mixed milk supply was contaminated by the owner of the suspected well adulterating the milk; 8 cases occurred in the house with the suspected well and 78 cases among contributors of milk to the creamery, and who, of course, were the largest consumers of their skimmed milk.	Dr. Schroder. Zeitschft. f. med. Beamte, Berlin, 1891, IV., pp. 227-262.
104	July, 1890.	Wyandotte, Mich	11	2	11	100	All supplied with milk from a stall-fed cow which drank water from a well polluted with animal matter. Cases continued to occur as long as this milk was used, and disappeared upon its stoppage, except in 1 family, who returned to the milk, and this was followed by 2 more virulent cases in the family. Professor Vaughan examined the milk and water bacteriologically and while failing to discover Eberth's germ, he found similar pathogenic germs in both media, in larger proportion in the cow's milk.	Dr. E. P. Christian. Am. Lancet, Detroit, 1891, n. s., XV., pp. 121-128; Phys. and Surg., Detroit, 1892, XIV., pp. 337-343.
105	August, 1890	Lauchstadt	74	9			The first and greatest number of cases occurred at a watering resort, which was supplied both with water and milk from a farm where typhoid cases had occurred.	Dr. Penkert. Zeitschft. für med. Beamte, Berlin, 1891, IV, p. 50.

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[1] Or more.

[2] Several deaths.

MILK EPIDEMICS—Continued.

TABLE I.—*Typhoid (Busey and Kober)*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Percent.	Circumstances of outbreak.	Reporter and reference.
106	August 1890	Waverley, Randwick, Sydney.	80		. . . . .	. .	These cases occurred in 80 households, and "the outbreak was clearly proved to be caused by contaminated milk." [We have been unable to refer to Dr. Thompson's original report.]	London Lancet, 1891, Vol. I, p. 225.
107	August 1890	Toorak, Australia.					A number of cases occurred, all pointing to a particular milk supply derived from a farm which was watered by a creek to which the cows had free access, an orchard on which infected night soil had been deposited drained into the creek higher up. No evidence given whether the milk had been adulterated with this polluted water, where the cans were washed, or whether udders were infected while cattle waded in the stream.	Austrl. Med. Jour., 1890, n. s., XII, p. 422.
108	September and October, 1890	Edinburgh. . . .	63	3		.	The outbreak occurred in 41 families who derived their milk from a farm where a case of typhoid was found, and 2 others subsequently occurred there. Sanitary condition bad, milk cans filled in a tainted atmosphere; water supply found to be contaminated by sewage and liable to gross pollution. Milk supply was stopped until a better water supply had been provided, after which no more cases occurred.	Dr. Harvey Littlejohn. Edinburgh Med. Jour., 1890-91, XXXVI, Part II, pp. 801-814; Brit. Med. Jour., 1890, Vol. II, p. 1318.
109	1891. . . .	United States. . .	2	. . . .	. . . .	. .	Dr. Brady describes 2 cases of typhoid fever which he attributed to infected milk, and considers it perfectly conceivable when we recall the sanitary condition of the average milk farm, and the dairy boy with bespattered boots, dirty hands and shirt, etc.	Dr. E. J. Brady. Cincinnati Lancet and Clinic, 1892, n. s., 28, p. 20.
110	1891.	Decatur, Ill.	5	. . . . .	5	100	Typhoid fever at dairy conveyed by digital infection, as dairy hands also assisted in nursing the typhoid patients.	Dr. E. J. Brown. Trans. Ill. Med. Society, Chicago, 1891, XLI, pp. 145-148.
111	February, 1891. . . .	Avondale.	12	. . . .	12	100	Two cases of typhoid fever at dairy. Milkers and dairy hands also assisted in nursing. Water probably contaminated, and owner in the habit of diluting the milk.	Dr. E. W. Mitchell. Cincinnati Lancet and Clinic, 1892, n. s., 28, p. 647.

112	June, 1891	Grosse Isle, Mich. . .	8	1	8	100	All these cases received the milk from 1 cow which had no access to pure water, but drank from a nearly dried-up swamp on the island. (No bacteriological examination of the water.)	Dr. E. P. Christian. Phys. and Surg., Detroit and Ann Arbor, 1892, XIV, 337-343.
113	August, 1891	Shawland, Glasgow.	42	4	37	89	Mild case of typhoid at the farm in August. Dung pit located near by received the typhoid excreta; the water supply contaminated from this dung pit; other unsanitary surroundings.	Dr. A. M. Campbell. Public Health, 1891-92, Vol. IV, p. 275.
114	October, 1891	Borough of Nanticoke, Pa.	42	. .	31	74	A case of typhoid fever at the dairy farm, attributed to a contaminated well which received drainage from a cemetery. (?)	Dr. L. H. Taylor. Annales Hygiene, Philadelphia, 1892, Vol. VII, p. 393-403.
115	Spring, 1892 .	Plymouth, England .	12	1	12	100	A fatal case of typhoid occurred at the milk farm twenty days before the present outbreak. The parents continued their dairy work while nursing their sick child.	Dr. F. M. Williams, M. O. H. Brit. Med. Jour., 1892, Vol. I, p. 1157.
116	August, 1892. . .	Springfield, Mass. .	150	25	101	67	After a painstaking investigation, traced to a particular milk farm, where cases of typhoid fever had occurred ever since last spring. Well liable to infection from dejecta of patients. Milk contaminated by placing cans in the well for the avowed purpose of keeping the milk cool.	Drs. Sedwick and Chapin. Boston Med. and Surg. Jour., CXXIX, 20, p. 485, 1893.
117	August 20 to September 10, 1892	Somerville, Mass.	35		30	86	Epidemic traced to a particular milk supply. The son of this milkman handled and delivered the milk while suffering from a mild attack of typhoid fever, which had remained unrecognized until the investigation disclosed exact facts.	Dr. W. T. Chapin. Boston Med. and Surg. Jour., CXXIX, 20, 1893, p. 485.
118	September 14 to October 15, 1892	Greenwich, Rotherhithe.	61	. .	56	91	This epidemic was limited to consumers of ice cream manufactured by Italian vender. Investigation revealed the existence of several cases of enteric fever in two ice-cream shops, and much reason for believing that the ice cream was prepared in dangerous proximity to the patients.	Dr. George Turner. Practit., London, 1892, XLIX, p. 141-160.
119	1893. .	Altenmuhr	[1]	. .		. . . .	A house epidemic existed at a certain farm. A young gentleman took sick with enteric fever while visiting a neighboring chateau. He had been supplied with milk from this farm, and his female servants who carried the milk were taken sick likewise.	Dr. Franz Spaet. Arch. fur Hygiene, Munchen and Leipzig, 1893, XVII, p. 306.

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[1] Several cases.

TABLE I.—*Typhoid (Busey and Kober)*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Percent.	Circumstances of outbreak.	Reporter and reference.
120	1893	Bandon.		. . .		. . . . .	In this instance Dr. Welphy traced the infection to a creamery which collected milk from a number of farms, at one of which a few cases of enteric fever occurred, infection originally carried from Cork. The milk at these creameries is separated, the cream made into butter, and the skimmed milk returned to farmer, thus causing intimate relationship, and the disease may easily become widespread among the users of the milk.	Dr. Welphy. Brit. Med. Jour., 1893, Vol. II, p. 698; London Lancet, 1894, Vol. II, p. 1085.
121	February, 1893	University of Virginia	14		14	100	These cases of a typical typhoid fever occurred among the students of the university, all boarding at the same hotel and consuming a particular milk supply from a dairy which is located on the banks of a creek which receives the sewage from one of the main university sewers. An ignorant negro, who lives 1 mile above the dairy, had typhoid fever during the preceding fall, and his dejecta was thrown on the ground without disinfection. The milkman used creek water to wash the udder of the cows.	Dr. Wm. C. Dabney. Med. News, Philadelphia, 1893, LXIII, 630-632.
122	May, 1893.	Oakland, Cal. . . .	302		228	70	These cases occurred within one month, and as 70 per cent were consumers of milk from one particular dairy, a sanitary inspection was made and revealed the following facts: A typhoid-fever house in close proximity, dejecta thrown on the ground close to a small dam in the creek, from which a pipe supplied a large tank 75 feet below with water for dairy purposes; moreover, this polluted water also flowed through the cow pasture.	Dr. S. M. Mouser. Occident. Med. Times, Sacramento, 1893, VII, pp. 503-504.
123	July, 1893.	Paisley, Renfrewshire	86	. .	86	100	This epidemic was traced to the consumption of ice cream made at the premises of a vender where an unreported case of typhoid fever was found, and this patient	Dr. Campbell Munro. Brit. Med. Jour., 1894, Vol. II, p. 829.

							had remained in contact with the business during most of her illness.	
124	August, 1893	Rostock	[1]				All traced to milk from a suburban dairy found in a most unsanitary condition; no privy, but a highly polluted well, which was used for washing the utensils and very likely also for adulteration.	Dr. Lesenberg, city physician, quoted by Dr. Dornblüth. Jahrbuch f. Kinder Krankheiten, 1893, XXXVI, p. 181.
125	July, 1893	Vicinity of Bethesda, Montgomery County, Md.	15	1	15	100	This limited epidemic was intimately connected with a certain milk farm, the owner of which was obliged to use a neighbor's well, in whose family typhoid fever had occurred during the summer of 1892; three weeks after using this well the first case occurred at the milk farm, and shortly afterwards the owner of the well was also taken sick. This resulted in a cleaning of the well, which was found to be contaminated with a very foul sediment, a dead chicken, and other organic refuse. The season being unusually dry, and the ground water being low, had resulted in concentration of the impurities, and as this well had been used for dairy purposes, it was doubtless the source of infection.	Unpublished memoranda furnished by our friend Dr. George Lloyd Magruder, of Washington, D. C., and Dr. W. F. Elgin, of Montgomery County, Md.
126	July to September, 1893.	Shildon, Durham Co.					In a very extensive epidemic of enteric fever a large share in spreading the fever was due to a particular dairy, where cases of typhoid fever existed and the wife who managed the milk business also nursed the sick children. There was, moreover, a direct connection between the sewer and the room in which the milk and utensils were kept.	Dr. Bruce R. Low. Report to the local government board on an outbreak of enteric fever at Shildon, London April 23, 1894.
127	1894.	Castle Island Ireland.					A serious outbreak was traced to a creamery receiving among others the milk from a farm where enteric fever had occurred, and which was handled by a person who also assisted in nursing those suffering from the disease. The cream had been separated and the skim distributed in due proportions among the different farms.	Brit. Med. Jour., 1894, Vol. I, p. 815.
128	January and February, 1894	Richmond Hill Surrey County.	55		52	94	Traced to a common milk supply; no evidence of typhoid fever at the milk farm, although the disease had prevailed in the vicinity; very unsanitary conditions, such as liquid and semiliquid filth surrounding 36 cows. The epidemic speedily subsided after stoppage of the milk supply from this dairy.	Drs. Rowland and Seaton. Brit. Med. Jour., 1894, Vol. I, p. 1325.

[1] Several cases.

MILK EPIDEMICS—Continued.

TABLE I. *Typhoid (Busey and Kober)*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Per cent.	Circumstances of outbreak.	Reporter and reference.
129	March and April, 1894	South Lambeth	59	10	55	93	Traced to a particular milk depot, affording no other evidence except unclean methods and a water supply subject to pollution from the yard drain. The water tank, on being emptied, contained a deposit of 4 inches of offensive matter; no bacteriological examination.	Brit. Med. Jour., 1894, vol. I, p. 1148.
130	March, 1894	Montclair, N. J.	107	14			Of 44 families supplied with milk from a particular dairy, typhoid fever occurred in 28, or 63.6 per cent. Of 20 cases reported from Bloomfield and Glenridge, 18 were traced to the same dairy, where a case of typhoid occurred February 11, but the sale of milk was not stopped until March 29, and epidemic checked promptly after that date. Unsanitary condition at dairy and polluted well water.	Dr. R. C. Newton. Med. Record, N. Y., 1894, XLV, pp. 713-715.
131	May, 1894	Brixton	60	10			This epidemic was traced to a milk farm where the cows were partially fed on fresh grass cut from the fields of a sewage farm, it was also shown that water from a brook running through the same land, and presumably contaminated, had been used to adulterate the milk.	Dr. Verdon, M. O. H. Brit. Med. Jour., 1894, Vol. 1, p. 1112.
132	August, 1894	Montclair, N. J.	19	1			Fourteen of these cases found in close proximity to a bakery where ice cream was sold and made in a very filthy place. A case of typhoid had occurred at this bakery, and persons who made the ice cream also assisted in nursing. Of 10 cases, 8 had used ice cream or milk from this bakery, and the disease was promptly checked upon closing the bakery.	Dr. Thomas Horton. Med. Rec., N. Y., 1894, XLVI, p. 651.
133	July, 1894	Bayhead, N. J.	15		15	100	This limited outbreak was confined to customers of a milk dealer who derived his supply from three dairies, at one of which a young man was taken sick July 1, with what proved to be a case of typhoid fever, and continued to milk his cows daily until July 11. The first case of typhoid fever among consumers of the milk occurred	Dr. W. H. Katzenbach. N. Y. Med. Record, 1895, vol. 47, p. 165.

							July 14, and the last case nineteen days after this patient stopped milking. How the infection could be conveyed may "be left to the imagination of those who are familiar with the personal habits of some who work on dairy farms."	
134	December, 1894	Arbroath, Scotland	44				This epidemic was traced to an unrecognized case at a dairy. The patient there was a woman 64 years old who had been waited on by two other women, who also milked the cows, washed the milk vessels, and attended generally to the sale of milk. Many of the later cases of the outbreak were not directly attributable to the milk sale, secondary centers of infection having, as is quite common, been established.	London Lancet, Vol. II, 1894, p. 1517.
135	January, 1895.	Great Harwood.	80	5		100	Consumers of raw milk were attacked more virulently and with greater certainty than those persons who took the milk in coffee or tea. The chief symptoms were headache, often diarrhea, sometimes nausea, characteristic temperature, and frequently abdominal rose spots. Traced to a milk farm, where a young woman who assisted in milking the cows and looked after cleaning the cans had been sick since and prior to the outbreak with what she thought to be a cold. Upon examination she was found with a coated tongue, a pulse of 108, temperature 100°, and a few days afterwards rose-colored spots appeared on her body—in fact, a typical case of ambulatory typhoid fever. The decline of the outbreak, allowance being made for the period of incubation, coincided with her withdrawal from the dairy operations.	Dr. Edward Sargeant, London Lancet, Vol. I, 1895, p. 1328; Brit. Med. Jour., Vol. I, 1895, p. 1110.
136	April and May, 1895.	Stamford, Conn.	307				Traced to the premises of a milkman whose barns were in the rear of his lot, surrounded on all sides by dwellings and outhouses; his tank for cooling milk was fed from a well 12½ feet deep and filled with water to within 1½ feet of the surface. West of the pump were two outhouses, one 20 feet and the other 15 feet, each above the level of the bottom of the well, and the drainage from these led directly toward the pump. It is believed that the typhoid germs were brought to this neighborhood by Italians who had been at work in the vicinity of the dairy and the disease was traced almost directly to their camp. The well water was examined by Dr. T. M. Prudden, and found to be swarming with bacteria	Med. Record, N. Y., vol. 47, pp. 562, 627.

MILK EPIDEMICS—Continued.

TABLE I. *Typhoid (Busey and Kober)*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Percent.	Circumstances of outbreak.	Reporter and reference.
157	April and May, 1895....	New Milford, Conn..	[illegible]	......	......	......	The daily papers contain accounts of an epidemic of typhoid fever which is prevailing in New Milford. The disease is said to have been distributed by milk obtained from a certain farm in the neighborhood. Up to May 9, 23 cases had been reported. Details wanting.	Med. Record, N. Y., vol. 47, p. 627.
158	June 22, 1895........	Woolwich...... ..	19	......	......	......	In 10 of these cases the milk was supplied from the same dairy and the others from various sources. In 4 cases the milk supply was from the Plumstead dairy, where the epidemic first broke out. This dairy has been closed by the authorities.	Brit. Med. Jour., Vol. I, 1895, p. 1423.

TABLE II. *Scarlatina (Hart).*

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Percent.	Circumstances of outbreak.	Reporter and reference.
1	June, 1870.	Penrith.	111	3	111	100	Scarlet fever at milkman's house. Cows milked by nurses. Milk kept in the back kitchen of the cottage.	Dr. M. W. Taylor. Brit. Med. Jour., Vol. II, 1870, p. 624.
2	June and July, 1870	St. Andrews	26	2	26	100	Milk boy had sore throat and peeling off of the skin while carrying milk. Other cases at farmer's house. Wife nursed, and milked the cows.	Prof. O. H. Bell. Brit. Med. Jour. Vol. II, 1870, p. 489.
3	1872	....	21	......	21	100	Twenty one children in a particular locality attacked within a fortnight, supplied with milk from a house where several children were ill of scarlatina.	Dr. M. K. Robinson, M. O. H. Annual Report of Medical Officer of Health, 1872.
4	June, 1875..	South Kensington. ..	19	......	19	100	This epidemic affected a number of guests and servants of a house who had partaken of cream in one form or other on June 9, 1875. First cases occurred between June 11 and 14. Mode of transmission unexplained.	Dr. Geo. Buchanan. Report of Medical Officer of local government board, No. VII, 1876, p. 72.
5	July, 1876.	Handsworth.........	37	4	37	100	Scarlet fever at dairy in the middle of June. Some of the milk and pans kept in the house; the dairy room communicated directly by a doorway with one of the living rooms.	Dr. T. B. Welch, M. O. H. Brit. Med. Jour., Vol. II, 1876, p. 225.

6	April and May, 1877.	New Barnet	140	....	131	93	The epidemic burst upon the district very suddenly, 128 cases occurring between April 29 and May 4; in addition to the scarlet fever cases there were 120 of sore throat. Dr. Saunders says that the cause of the epidemic must be regarded as an accident	Dr. C. E. Saunders, M. O. H, Sanitary Record, Vol, VII. 1877. p. 69.
7	June, 1878	High Ashurst and Headley.	20	2	...	.	First cases in the children of a cowman, who nursed the patients and milked the cows, though he did not himself have the fever and the milk was not taken into his cottage	Dr. E. L. Jacob, M. O. H. Brit. Med Jour., Vol. I, 1880, p. 139.
8	May and June, 1879.	Westgate, Newcastle-on-Tyne.	23	10	..		This very malignant epidemic was traced to a particular dairy where no scarlet fever existed, although prevalent in close proximity. The surroundings favored directly or indirectly the contamination of the milk with organic matter which frequently smelled offensively	Dr. H. E. Armstrong, M. O. H. Brit. Med. Jour., Vol. II, 1880, p. 671.
9	August, 1879.	Fallowfield, near Manchester.	35	... ...	35	100	Twenty-four of the cases occurred within a space of thirty-six hours; no scarlet fever at farm or dairy and no disease among the cows. One of the milkers lodged, however, where his grandchild was lying in the full height of desquamation after scarlet fever.	Dr. B. Airy. Brit. Med Jour., Vol. I, 1880, p. 107.
10	July and September 1880	Paddington and Bayswater	84	.			Scarlet fever at milk farm among the children of the milkers, there were two distinct outbreaks, one at the end of July, the other at the end of September	Dr J. Stevenson, M. O. H. Brit Med. Jour., Vol. II, 1880, pp 596, 632.
11	October, 1880	Dundee					No case at farm, but female servant visited a house and contracted the disease, which was communicated to a person connected with another dairy	Brit Med. Jour., Vol. II, 1880, p 790
12	November, 1880, and January, 1881	Ilkley, Yorkshire	10 4		10 8	100 88	Infection probably conveyed from a fever house visited by the dairyman, who was in the habit of taking his milk can into the houses	Dr T. Scott, Brit Med. Jour., Vol I, 1881, p 604.
13	January, 1881	Bromley, Kent	.				This sudden outbreak affected 18 families, supplied from a particular dairy, where one of the employees continued at work while 4 of his family had scarlet fever at home	Dr C. O. Baylis, M. O. H. Brit. Med. Jour., Vol I, 1881 p. 14
14	January, 1881	Halifax.	40	86			The farmer's man who milked the cows and brought the milk to the customers in Halifax had 4 children ill of scarlet fever, and probably helped to nurse them	Drs Ankey and E. Ballard. Brit Med. Jour., Vol. I, 1881, p. 255, Vol. II, 1881, p 185
15	April, 1881	Keswick	.	.	.		The dairy adjoined a house where scarlet fever had existed for several weeks. Cows milked into an open can, which was carried across an open yard past the affected house, 30 to 40 families being affected	Dr J. Robertson M. O. H

[3] Families.

MILK EPIDEMICS—Continued.

TABLE II. *Scarlatina (Busey and Kober)*.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Per cent.	Circumstances of outbreak.	Reporter and reference.
16	January, 1882.	Greenock.	20		20	100	All traced between January 19 and February 1 to a milk supply derived from a farmhouse where 4 children were sick with scarlet fever. The milk supply was at once stopped.	Dr. Wallace, Brit. Med. Jour., 1882, Vol. III, p. 457.
17	January 14, 1882. . .	Charing Cross	1				One of the dairy hands in London first showed symptoms of sore throat January 14th and on the following day one of his children developed scarlatinal rash. There was no scarlet fever at the farm or for miles around, but Dr. Klein ascertained that a cow calved about the early part of January, and on February 1 (date of his investigation) he noted that she had here and there lost portions of her coat and that her buttocks and posterior udder were foul and stained by excremental matter, and concluded that he had to deal, very likely, with puerperal scarlatinal infection from the cow, as puerperal fever in women was not infrequently due to scarlatinal infection.	Dr. W. H. Power, Report Medical Officer Local Government Board, 1882, No. XII, pp. 63–71.
18	January 14, 1882 . . .	Camberwell. . . .	39	. . . . .	32	82	Milk supplied from above dairy. . . .	Do.
19	January, 1882	St. Giles, St. Pancras, Marylebone	32		32	100	. . do.	Do.
20	January, 1882.	Bloomsbury. .	.			.	Outbreak connected with a particular milk supply.	Mr. W. H. Power, quoted by Prof. A. M. Davies, Prov. Med. Jour., Leicester, 1889, Vol. VIII, p. 587.
21	February and March, 1882.	Oxford. . . .	29	. . . . . .	29	100	This epidemic was traced to a particular milk supply, and the cases really consisted of 10 of scarlet fever, 18 of sore throat, and 1 of diphtheria. Investigation showed that a case of scarlet fever occurred at dairy February 27, and that this case had been preceded by a case of diphtheria next door to the dairy.	Dr. S. D. Darbishire, St. Barth. Hosp. Reports, XX, 1884, pp. 93–100.
22	October and November, 1882.	Greenock. . .	47	. . . . . .	12	25	The first case occurred in the person of a milk dealer who took sick with what he considered an ordinary cold on October 6. One of his children became affected about eight days after; no medical man was called in to see these first cases.	Dr. Wallace, M. O. H., Brit. Med. Jour., 1882, Vol. II, p. 1325.

23	July, 1882	Sunderland					Mr. Harris accidentally discovered the existence of scarlet fever in a dairy, and upon inquiry among people in whose homes deaths from the disease had occurred, it was shown that in four such instances the people had been supplied with milk from the infected source. Mr. Harris was unable to discover how many nonfatal cases owed their origin to this source.	Brit. Med. Jour., 1882, Vol. II, p. 100.
24	April, 1883	Wolborough					Scarlet fever at milk seller's house. Of 16 families attacked, 9 were supplied with the suspected milk.	Brit. Med. Jour., 1883, Vol. I, April 21, 1883.
25	October, 1883	Dundee	17		17	100	Epidemic traced to a farm where there was a boy sick with scarlet fever, and his nurse also milked and handled the milk in question.	Lancet, London, 1883, Vol. II, p. 696, Brit. Med. Jour., 1883, Vol. II, p. 839.
26	August, 1884	do.					A large number traced to a milk farm where one of the boys employed suffered from the disease but continued at work. Milk supply stopped and epidemic checked.	Brit. Med. Jour., 1884, Vol. II, p. 433.
27	October, 1884	Greenock					An extended outbreak involving some 30 families mostly supplied from one source. The sanitary conditions at dairy warranted the stoppage of the milk.	Brit. Med. Jour., 1884, Vol. II, p. 924.
28	January, 1885	Paisley					Scarlet fever is reported as exceedingly prevalent in one district of the town. Milk taken from a dairy where one of the children was suffering from the disease.	Brit. Med. Jour., 1885, Vol. I, p. 41
29	May and June, 1885	Rostock	8		8	100	Directly traced to a milk farm where scarlet fever prevailed, and convalescents assisted in milking.	Dr. T. Dornbluth, Jahrbuch f. Kinderkrankheiten, Leipzig, 1893, XXXVI, p. 174-191.
30	November and December, 1885	Marylebone and St. Pancras					This epidemic is believed to have been directly contracted from a peculiar disease affecting the cows, and which Dr. Klein believed to be identical with human scarlatina, and he isolated a micrococcus. Hendon disease.	Dr. A. W. Blyth, Brit. Med. Jour., 1886, Vol. I, 223. W. H. Power, Report Medical Officer local government board, 1885, pp. 73-84.
31	December, 1885	Dorset Square	60		60	100	All patients had consumed milk from the Hendon dairy. Persons who drank only boiled milk were spared.	Dr. A. W. Blyth, Brit. Med. Jour., 1886, Vol. I, p. 223.
32	1886	St. George, Hanover Square					A limited group of cases supposed to have been due to the consumption of condensed milk in which Klein found an organism, which he considered characteristic.	Dr. Corfield, Brit. Med. Jour., Vol. II, September 22.
33	June, 1886	Liverpool					Several cases traced to a particular milk supply, and believed to be due to disease of the cow.	Dr. J. M. Howie, Brit. Med. Jour., 1886, Vol. I, p. 1231.
34	December, 1886, and January, 1887.	South Wimbledon and Merton	635	4	577	91	The outbreak was extensive and affected as many as 119 patients one day, mostly all consumers of a particular milk supply. 29 cases occurred among patients supplied from a different dairy, but the cows had been in contact with those supplying dairy No. 1. Attributed to a communicable disease of the udder.	Dr. C. H. Cooper, Trans. Epid. Soc., London, 1888-89, n. s., VIII, 38-47. W. H. Power, Report Medical Officer local government board, 1886, pp. 327-339. Sanitary Record, London, 1888-89, X, 422-426.

MILK EPIDEMICS—Continued.

TABLE II.—*Scarlatina Rasey and Kober*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Percent.	Circumstances of outbreak.	Reporter and reference.
35	April, 1887	Toxteth Park, Liverpool	..		....		This outbreak was traced to the milk supply of a certain dairy, but investigation failed to reveal the presence of human scarlet fever or contamination from that source, and no explanation except that a cow had calved there about April 1. Dr. Steeves also reported that cases of diphtheria appeared about the same time and localities as scarlet fever.	Dr. G. W. Steeves, Brit. Med. Jour., 1888, Vol. II, p. 911.
36	August and September, 1887	Dundee	783 813	20	....	.	The Medical Officer reported to the sanitary committee of the Dundee police commission the outbreak of scarlet fever in connection with a number of dairy premises in the parish of Murroes, from which milk was sent into Dundee.	Brit. Med. Jour., 1887, Vol. II, pp. 783-786.
37	November, 1887	Hyde Park, Chicago	..	.	.	..	Scarlet fever appeared among the children of four families supplied by a milkman whose child was sick with "acute inflammation of the kidneys;" no scarlet fever card had been posted on the house, nor did the milkman cease delivering milk.	Sanitary Record, London, 1887-88, n. s., Vol. IX, p. 478.
38	December, 1887......	Great Britain.. ..	5		5	100	Traced to a milk supply derived from a diseased cow whose milk was of grayish color and later the animal's skin desquamated freely.	Dr. H. Mallins, London Lancet, 1888, Vol. I, p. 119.
39	December, 1887......	Cannes......	(?)	....	..		Traced to a dairy where all the cows were suffering from a diseased condition of the udders; they saw no ulceration, but the teats were covered with large scabs, and the dairyman stated that the scabs had been preceded by a vesiculo-pustular eruption, which broke while milking. The outbreak was limited to consumers of milk from these cows. (No sores on the hands of milkers.)	Dr. H. Blanc, Lancet, London, 1888, Vol. I, p. 545.
40	January and February, 1888	Newcastle.... ..	19	......	19	100	Traced to a milk farm where scarlet fever had occurred several months previously, and the cows presented evidences of having been recently sick, such as denuded hair, etc.	Dr. H. E. Armstrong, Public Health, May and October, 1888.

41	March 15 to April, 1888	Garnethill, Glasgow	96	3	96	100	Traced to consumers of a particular milk supply; no scarlet-fever cases at dairy or milk farm to account for outbreak. One of the cows was found thin and mangy looking, casting hair generally; several sores on teats covered with bloody scabs. A calf fed on this milk seized with a high fever which nearly killed it, but from which it is now recovering, with loss of hair and copious casting of the skin.	Dr. J. B. Russell, M. O. H. Sanitary Journal, Glasgow, 1888–89, n. s., XII, pp. 70–74.
42	June, 1888	Pollokshiels, Glasgow	70				Traced to a particular milk farm where scarlet fever prevailed. A milkmaid was one of the victims, and while sick continued at work.	Dr. Carmichael. Brit. Med. Jour., 1888, Vol. II, p. 32; Lancet, London, 1888, Vol. II, p. 179.
43	July 2–14, 1888	Newcastle	116				These cases occurred in 63 families supplied by the same dairyman, and all took sick within a week with scarlet fever, sore throat, etc. Three of the children at milk farm exhibited certain symptoms of redness of the throat, tonsils, and fauces, but no signs of desquamation.	Dr. H. E. Armstrong. Sanitary Record, London, 1888–89, n. s., X, p. 64.
44	August, 1888	Newcastle	71		61	82	Milk derived from a farm where there was throat disease in the family of one of the dairy helpers; there were also 5 out of 24 cases of diphtheria among drinkers of the same milk.	Dr. H. E. Armstrong. Public Health, September, 1888, quoted, by Dr. A. M. Davies, ibid.
45	September and October, 1888.	Garnethill Park epidemic, (Glasgow).	56				Traced to a particular milk supply distributed from a shop where cases of scarlet fever and sore throat were found.	Dr. J. B. Russell. Sanitary Journal, Glasgow, 1888–89, n. s., XII, pp. 268–272; Lancet, London, 1888, Vol. II, 1079.
46	October, 1888	Spennymoor, Durham County.	83	15			Attributed to a contaminated milk supply (doubtful evidence).	Dr. David Page. Report to local government board, London, 1889, p. 9.
47	October, 1888	Greenock	55				The majority of cases occurred among the well-to-do customers of a particular milk seller, who evidently had an infected dairy and quite innocently sent out the infected milk	Brit. Med. Jour., 1888, Vol. II, p. 956.
48	December, 1888	Govan.	57		55	94	Of 74 families supplied with the suspected milk the disease appeared in 23 families and furnished 35 cases, while there were only 2 cases among 605 families supplied from other dairies. Origin not determined.	Sanitary Record, February, 1889.
49	December, 1888.	Ibrox and Paisley road	65		35	100	Traced to a milk shop, where there was a case of scarlet fever in the son of the keeper, whose family occupied the back kitchen. Shop promptly closed.	Dr. Geo. McKay. Sanitary Journal, 1888–89, XII, p. 341
50	January, 1889.	Hillhead and Patrick, Glasgow					A considerable number of cases have occurred within pistol shot of one another and attributed to infected milk.	Brit. Med. Jour., 1889, Vol. I, p. 34.

¹ Several cases.

TABLE II—*Scarlatina (Busey and Kober)*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Per cent.	Circumstances of outbreak.	Reporter and reference.
51	February, 1889	Macclesfield [illegible]	47 10		47 10	100 100	All traced to a particular milk supply believed to be infected by a diseased cow among the dairy stock.	Dr H. F. Parsons. Report Medical Officer local government board, 1889, pp. 89-114.
52	October, 1889	Kilmarnock	12				Milk derived from a dairy where 2 cases of scarlet fever had occurred.	Dr J. C. McVail. Sanitary Journal, Glasgow, 1890-91, n. s., XIV, pp. 73-75.
53	March, 1890.	Brewster, Putnam County, N. Y.	24		24	100	The daughter of a dairyman was taken with scarlet fever the day after her arrival in New York City. Two weeks after her recovery she returned home, two weeks later her youngest sister, who slept with her, presented evidence of the disease, followed three weeks later by a number of cases in the village. The milkman had washed and wiped his cans with white flannel cloths left in the barn by a peddler of rags, which were probably the cause of first infection.	Dr L. H. Miller. Med. Record, N. Y., 1890, XXXVII, p. 587.
54	June, 1890.	Crosby, Liverpool	30		30	100	An explosive outbreak traced to a milk farm where there was no other evidence except a sick milch cow, pronounced by the veterinarian to be suffering from "bovine tuberculosis."	Mr Limrick, M. O. H. Lancet, London, 1890, Vol. I, p. 1315.
55	January, 1891.	Edinburgh					In this epidemic of scarlet fever a large number of adults were attacked in proportion to children. Adults partook of the poison in the form of cream in tea, which seems in certain cases to increase the virulence of the poison.	Dr Harvey Littlejohn. Lancet, London, 1891, Vol. I, p. 109.
56	January, 1891.	Bristol	250	15		..	These cases were all traced to two milk rounds and two dairy farms, located in a fever district, and infection possibly conveyed by workmen who lived in infected localities. No scarlet fever at farms.	Dr S. S. Davies, M. O. H. Public Health, 1891-92, vol. 4, p. 362.
57	1891	England			....	.	A limited outbreak of scarlet fever, which to myself and two other medical men appeared to be due to milk. Preceding the outbreak one of the cows at the suspected dairy had had an eruption on the udder, but almost by	Dr W. N. Thursfield. Public Health, 1891-92, vol. 4, p. 133.

							accident I discovered that a boy not residing at the dairy and who had casually been employed to milk had, just previous to the outbreak and when milking, suffered from a slight sore throat. * * *	
58	February, 1891	Cardworth	5				Traced to a particular milk derived from a farm where one of the dairy hands was convalescing from scarlet fever.	Dr. B. Hill. Public Health, London, 1890-91, Vol. III, p. 487.
59	March, 1891	Manor Hill and Clifton road, Sutton, Coldfield	40	5	30	75	Traced to a milk farm with no other evidence except sickness among dairy stock, indications of recent ulceration on the teats, and progressive emaciation. Milk supply stopped and, with exception of one or two sporadic cases, no further cases arose in that part of the borough	Dr. B. Hill. Public Health, London, 1890-91, Vol. III, pp. 487-491; Brit. Med. Jour., 1891, Vol. II, p.
60	October, 1891	Whitchurch	[1]				This outbreak was traced to a dairy where a case of scarlet fever had occurred and, as convalescent, was prematurely permitted to handle milk and utensils	Dr. Pritchard. Brit. Med. Jour., 1891, Vol. II, p. 1179
61	November, 1891	Bushhill Park	33		33	100	This sudden outbreak was traced to a common milk supply, no evidence of human infection, but cows suffered from an eruption on udder	Dr. S. M. Copeman. Report Medical Officer local government board, 1891-92, XXI, pp. 69-78.
62	March and April, 1892	Charlton, S.E. London	57				Epidemic traced to a common milk supply from a farm at which no trace of infection could be found, except scabs and excoriations of the udder and teats among cows	Dr. Hamer. Public Health, 1891-92, vol. 4, p. 306.
63	March and April, 1892	Upper Clapton	145				Outbreak connected with a particular milk supply traced to a dairy where the child of one of the employees had scarlet fever	Dr. J. King Warry. Practitioner, London, 1892, XLIX, pp. 65-7.
64	April, 1892	Handsworth	143	1	89	62	Traced to a certain milk supply, but contamination of the milk not explained, unless due to a mild cow infection or to a polluted water supply	Dr. J. B. Welch. Public Health, London, 1892-93, V, 76-78.
65	May and June, 1892	Aston Manor	97		62	64	Connected with a certain milk supply, but infection not explained, unless of a bovine origin	Dr. Henry May. Public Health, London, 1892-93, Vol. V, p. 79.
66	August, 1892	Glasgow	224				This epidemic affected members of 94 families who obtained their milk from a certain farm where there was no evidence of scarlet fever, but an epidemic teat eruption among the milch cows	Drs. J. B. Russell and Arch. K. Chalmers. Glasgow Med. Jour., 1893, XXXIX, pp. 1-22.
67	September, 1892	Leyton, Essex					Most of the cases confined to customers of a certain milkman, who derived his supply from a farm where scarlet fever prevailed	London Lancet, 1892, Vol. II, p. 735.
68	November, 1893	Hastings					This limited epidemic invaded 26 houses, 18 of which had been supplied with the same milk. No scarlet fever at milk farm, but the cattle "were found to be all more or less suffering from febrile disturbance."	Dr. Scarlyn Wilson. Brit. Med. Jour., 1894, Vol. I, p. 815.

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[1] Several cases.

MILK EPIDEMICS—Continued.

TABLE II.—*Scarlatina—Busey and Kober*—Continued.

No.	Date	Place	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Per cent.	Circumstances of outbreak.	Reporter and reference.
69	December, 1893	Glasgow	30		28	93	This epidemic was traced to the milk from two dairy farms. At one of these a boy simply suffered from sore throat early in December which induced scarlet fever in others. At the other farm some of the dairy employees developed scarlet fever and had kept at work for one or more days after illness.	Dr. A. K. Chalmers. Brit. Med. Jour., 1894, Vol. I, p. 426, Glasgow Med. Jour., 1894, XLI, pp. 117-127.
70	March, 1894	Blackheath	89	1	..		The epidemic invaded families served by the same milk dealer. One of the employees had scarlet fever, unknown to him at first, and actually went about delivering milk while the rash was out upon him. Strong suspicion that the disease originated from the cattle.	Dr. Shirley F. Murphy. The Lancet, 1894, Vol. II, pp. 449, 910.
71	April, 1894	Patrick					This limited epidemic was traced to a milk farm in Dumbartonshire which supplied Patrick with the milk, and several cases of scarlet fever had occurred there.	Lancet, London, Vol. I, 1894, p. 1101.
72	October, 1894	Shirley, Warren, Southampton.	9	.	9	100	These cases were traced to a dairy where there had been an unrecognized case of scarlet fever. In a cottage, under the same roof as the milk, was found a case in the "desquamative" stage.	Dr. George H. Weston. Brit. Med. Jour., 1894, Vol. II, pp. 956-1408.
73	February, 1895	Hornsey sanitary district.	233	..	.		Certain of the farms from which the dairy in Hornsey drew its milk supply were either in or close to a village in which scarlet fever had been prevalent, though in a very mild form, since the summer of last year, so that the school at Hatton, the village in question, was closed, and it was found that at one of the milk farms the milk was actually under the same roof with a case of scarlet fever which occurred there January 8. As has been observed in previous epidemics of scarlet fever disseminated by milk, the type of the disease appears to have been mild, as no death from the disease has been reported.	Brit. Med. Jour., Vol. I, 1895, p. 550.
74	April, 1895	Hornsey			.	..	The outbreak of scarlet fever in North London just referred to has been followed by a smaller one, which, from the remarks of the chairman of the Hornsey district council, appears	Brit. Med. Jour., Vol. I, 1895, p. 772.

							to have been traced to one dairy. It was stated that the milk was distributed by two men, and that at the houses at which one of these men delivered milk there were no cases at all, while at those at which the second man delivered there were 15 or 16 cases of scarlet fever. At this man's house a child had suffered from scarlet fever, showing clearly the manner of infection as having taken place by infected clothing, and the germs were most likely conveyed into the milk while this man pushed his arm into the big can to fill the little ones during his rounds.	

TABLE III *Diphtheria (Hart).*

1	June, 1877.	Sutton, Surrey	15	......	......	.	No sickness at farm among men or beasts. The outbreak affected within 3 days 15 persons in 11 households, in good sanitary surroundings, and all supplied from 1 dairy.	Dr. E. L. Jacob, M. O. H. Brit. Med. Jour., Vol. II, 1879, p. 740.
2	May, 1878.	North London (Kilburn and St. Johns Wood, etc.).	264	38		...	No diphtheria or sore throat at milk farm, but Mr. Power subsequently raised the question whether garget in cows might not induce such changes in the milk as to give diphtheria to the human subject.	Dr. W. H. Power. Brit. Med. Jour., Vol. I, 1879, pp. 48-58. Trans. Pathol. Society for London, 1879.
3	July, 1878.	Weybridge, Surrey	60	2	60	100	Cases occurred simultaneously at milk farm and customers. Well water of dairy contaminated with sewage matter. Insanitary drains in the yard where milk cans were cleansed, and in the floor of the dairy was a gully leading to an unventilated drain.	Dr. E. L. Jacob, M. O. H. Brit. Med. Jour., Vol. II, 1879, p. 739.
4	August and September, 1878.	Leatherhead	55	5		...	No cases of diphtheria at milk farm or dairy; no sickness among the cows. The water used at dairy had at times smelled badly, and the pipe from the sink where milk cans were cleansed was not properly trapped.	Dr. E. L. Jacob, M. O. H. Brit. Med. Jour., Vol. II, 1879, p. 740.
5	October, 1878.	Addlestone, Surrey	48	.	...	...	No diphtheria at farm. Water supply very impure, and one of the cows had lately suffered with garget.	Dr. E. L. Jacob, M. O. H. Brit. Med. Jour., Vol. II, 1879, p. 739.
6	September, 1879.	Little Horton, Bradford.	7	1	7	100	A child had been ill with sore throat at farm about August 18, and another found with unmistakable diphtheria taken sick August 31; unsanitary conditions, dirty milk cans, washed over a sink, beneath which was found a chamber containing excreta, washtub near milk supply.	Dr. H. Butterfield, M. O. H. Brit. Med. Jour., Vol. I, 1880, p. 953.
7	December, 1880.	Surbiton	42	8	.	...	Traced to a particular milk supply where no cases of diphtheria existed at farm; in fact, no evidence of infection by human agency.	Dr. O. Coleman, M. O. H. Brit. Med. Jour., Vol. I, 1881, p. 140.

MILK EPIDEMICS Continued.

TABLE III. —*Diphtheria (Busey and Kober).*

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Percent.	Circumstances of outbreak.	Reporter and reference.
8	December 21-30, 1882	Devonport	31	5	27	93	Most of the persons attacked were liable to sore throats and many were adults (20 cases). Origin obscure; no evidence of diphtheria at dairy, but the surroundings were very unsanitary, and according to Dr. Parsons the infectious matter may have gained access to the milk by wiping out the cans with cloths which had been hung up in the narrow and close back yard and attracted impurities from the atmosphere.	Dr. Parsons. Report Medical Officer local government board, 1883, pp. 49-53.
9	January, 1883	Hendon Ward, Hendon urban sanitary district	62	5		...	This epidemic was traced to a milk supply derived from a dairy where the utensils were washed in a brook contaminated by sewage, and the milk was quite ropy and stringy. Evidence of previous cases in the vicinity. No apparent disease among the cows.	Dr. W. H. Power. Report Medical Officer local government board, 1883, pp. 42-48.
10	1883	Cardiff	...	......		...	This epidemic was traced to milk derived from a particular farm where diphtheria prevailed. The well was liable to sewage contamination, and dairy utensils were washed in this water.	Dr. Paine. Brit. Med. Jour., 1883, Vol. I, 973.
11	April, 1883	Putney	17	5	17	100	Out of 400 families supplied with the suspected milk the disease appeared in only nine. Diphtheria had prevailed in the district. No evidence of diphtheria at the farm (doubtful).	Drs. Walker and Blaxall. Sanitary Record, 1882-83, pp. 515-560.
12	June, 1886	Melrose, Mass	23	...	14	....	Epidemic traced to milk derived from a farm where diphtheria was known to exist.	Dr. Jul. S. Clark. Boston Med. and Surg. Jour., vol. 117, 1887, p. 100.
		Malden, Mass	27	...		...	Of these 27 cases 24 were supplied with milk coming direct from families in which the disease was known to exist. Of the 14 deaths 13 were in families using the milk known to be infected, and one in the family in which no history is had.	
13	July 11-18, 1886	Canterbury	231	......	......	......	All these cases were taken sick within one week; all had a local throat affection with white patches on tonsils and enlarged cervical glands, no fatal cases; many of the attacks were mild, some were more severe and lasted a fortnight; those were pronounced diph-	Dr. Wacher. Brit. Med. Jour., 1886, Vol. II, p. 397.

							theria the others were of a diphtheritic type. The milkman and family were suffering about the same time from sore throat; also the pet lamb. Cows apparently healthy, although four calves had recently suffered from diarrhea.	
14	October, 1886	Camberly and Yorktown.	135	16	124	92	The outbreak affected, especially, well-to-do families, who bought larger quantities of milk and stored for use. The result of the investigation at the milk farm proved negative, one of the milk carriers had scarlet fever in July, another developed the disease after the main outbreak; no disease among cows, except slight sign of "chaps" on their teats.	Dr. W. H. Power Report Medical Officer local government board, 1886, pp. 311-326.
15	October and November, 1886.	Military college near Camberly.	88	18			Traced to a common milk supply where polluted water had been used to dilute the milk.	Dr. Alf. T. C. Clark, brigade surgeon. Army med. dept Reports, London, 1887, XXVII, pp. 433 441.
16	January, 1887	Ealing	30	12	28	93	Outbreak sudden and affecting within one week customers of a particular dairy. Before and after this outbreak numerous cases of sore throat had been observed in families supplied from the same dairy, no evidence of disease among men or animals at the farm to account for the infection of the milk.	Dr. W. H. Power. Report Medical Officer, local government board, 1887, pp. 93 101.
17	November [illegible] to December 21, 1[illegible].	Enfield	213	48	179	84	This epidemic affected families residing in the best part of Enfield as well as persons living in less favorable localities, and seized persons supplied with a particular milk, however wide apart their residence. No evidence of diphtheria at dairy, but report refers to the unusual number of dead cats found in December and January in the vicinity.	Dr. Bruce Low. London Lancet, 1888, Vol. I, p. 1151.
18	January, 1888.	Oakleigh police station					This epidemic was supposed to have originated in the milk of a diseased cow, which had been used by the inmates of the station.	
19	January, 1888.	Princess Mary's village homes.	48				The water supply of the farms which supplied milk to the homes was found to be impure. One of the cows had "garget," and the epidemic began to decline rapidly eight days after the stoppage of the milk supply.	Dr. B. B. Loughead. Report board of health, Ohio, 1886-87, Columbus, 1888, II, p. 362.
20	1889.	East Kent					Dr. Robinson details several local outbreaks connected with the milk supply. At one farmhouse the disease was concurrent with a disease among the farm stock, and on the occasion of a previous outbreak at the same house the cattle were also concurrently affected. In another instance the first case was in a boy who had been feeding a dog with the carcass of a diseased cow. In the last sudden and explosive outbreak the disease was confined to consumers of milk derived from a farm where three cows had been sick.	Dr. Robinson. Brit. Med. Jour., 1889, Vol. I, p. 1247.

MILK EPIDEMICS—Continued.

TABLE III.—*Diphtheria (Busey and Kober)*—Continued.

No.	Date.	Place.	Number of cases.	Number of deaths.	Number of cases among milk consumers.	Per cent.	Circumstances of outbreak.	Reporter and reference.
21	January and February, 1889	Macclesfield and Upton	85	..	..	..	These cases occurred in 58 families, consumers of milk from a certain farm; severest cases occurred among the largest milk drinkers. Mode of infection obscure, but believed to be due to a diseased cow among the dairy stock. The epidemic ceased upon stoppage of the milk. Upon resuming to milk a suspected cow, new cases of scarlet fever and throat affections developed.	Dr. Parsons. Report of Medical Officer local government board, 1889, pp. 89-114.
22	June 1889.	Faelkes farm, Holbach medical district.	73	.	3	....	Traced to a dairy; infection probably contained in the milk sold June 9 and 10, milk farm in a most unsanitary condition and disease among milch cows.	Dr. H. Møller and N. Flindt. Ugeskrift for Læger, Kjobenhavn, 21, 4 R., 1890, XXI, pp. 405-410.
23	March, April, 1890	Glasgow	80	......	3	......	This epidemic was traced to a milk farm, where one of the dairymaids suffered from a sore throat of an erysipelatous character. The epidemic manifested itself chiefly in the form of severe sore throat, but in a number of cases a typical erysipelas developed. This conjunction of sore throat and erysipelas is interesting; and it may almost be said, from the acute character of the inflammation of the throat and its suppurative nature in some cases, that it also had many of the features of erysipelas. "In all, the tonsils and fauces were intensely congested, one having a distinct erysipelatous appearance. In several, the pharynx was also involved in the congestion. In two of those who drank freely of the milk and continued its use longest, there was membranous exudation in the shape of patches on the lips and tongue, but none on the tonsils and pharynx. Submaxillary glands were invariably enlarged, temperature was high during the first few days. Prostration was a marked feature."	Dr. Wm. Hunter. Glasgow Med. Jour., 1890, XXXIV, pp. 241-258.
24	October and November, 1890.	Croydon	100	....	......	....	Milk supplied by two dairies; Dr. Klein found at one of the farms ulcers in the teats of certain milch cows; supply from this farm stopped November 3, and only a few more	Dr. Carpenter. Sanitary Record, London, 1890-91, XII, p. 274 Dr. Philpot, Brit. Med. Jour., 1891, Vol. 1, p. 476.

							cases occurred afterwards. Dr. Carpenter thinks that the milk was contaminated during distribution, not at its source in the cow's udder.	
25	November, 1891.	Worcestershire	6	......	6	100	This house epidemic affected residents of the dairy farm all consumers of unboiled milk derived from cows suffering from a "febrile and eruptive epizootic."	Dr. W. N. Thursfield. Public Health, London, 1891-92, IV, pp. 130-134.
26	December, 1890	Surbiton, urban sanitary district.	27	3	27	100	Epidemic among customers of a particular milk farm, located in an infected district; the disease was promptly checked upon stoppage of the milk supply.	Dr. Coleman, M. O. H. Public Health, 1891-92, Vol. IV, p. 159.
27	July, 1893.	Hightstown, N. J.	28	11	28	100	These cases occurred within one week, and were all traced to a milk supply derived from a farm where a German boy assisted in milking while he had diphtheria.	Dr. T. B. Applegett. Medical News, 1893, vol. 63, p. 238.
28	December, 1894.	Ashtabula, Ohio	111	23	100	90	Traced to a particular dairy where one of the drivers of the milk wagons was suffering from sore throat as early as December 2, and perhaps a few days earlier. The first case among the infected families occurred December 4. Of the 111 cases and 23 deaths, 100, with 21 deaths, were in families that used this particular milk. Sale of milk prohibited December 11. Evidence pointing to infected milk is entirely circumstantial, since after repeated examinations the bacillus of diphtheria was at no time found in cultures from the milk or from the throat of the driver.	Report of Ohio State Board of Health, 1894, p. 298. Dr. Hopkins and Dr. Wm. T. Miller. The Western Reserve Medical Journal, April, 1895.

3. THE MILK SUPPLY OF CITIES IN RELATION TO THE EPIDEMIOLOGY OF TYPHOID FEVER.

THE MILK SUPPLY OF CITIES IN RELATION TO THE EPIDEMIOLOGY OF TYPHOID FEVER.

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Milk is a favorable culture medium for the typhoid bacillus. Therefore, if a small particle of matter containing this organism is introduced into milk the organism may undergo rapid multiplication and become disseminated throughout the bulk of the milk. The temperature at which the milk is kept and the number and kind of other bacteria present affect the rate at which the multiplication of typhoid bacilli takes place, and in some instances, no doubt, the bacilli, after gaining access to a body of milk, die out before that milk is drunk. In the majority of instances, however, it is probable that the bacilli will survive and so endanger all susceptible persons into whose alimentary canals such milk is taken.

Considering the tremendous multiplication which the bacilli can undergo within twenty-four hours in milk it is easy to appreciate how one bottle or can of infected milk taken into a dairy and there mixed with a large volume of milk may be responsible for exposure to infection of several thousand persons.

Dairy products, such as ice cream, buttermilk, butter and cheese, etc., made from infected milk may be factors in the spread of typhoid fever.

Ice cream.—It has been proven experimentally that the process of freezing does not at once destroy all typhoid bacilli, and outbreaks of typhoid fever have been traced quite definitely to infected ice cream.

Butter and cheese.—Butter made from milk experimentally infected with typhoid bacilli may retain the bacilli, according to Bruck,[a] as long as twenty-seven days, and according to Washburn[b] for as long as sixty days or more. Although it would seem, under ordinary circumstances, that the presence of many vigorous saprophytes, the washing out of large numbers of bacteria in the buttermilk, and the salting would lessen the chances of typhoid bacilli remaining in the butter, yet in view of the experimental evidence this dairy product should be kept in mind as a possible factor in the spread of typhoid fever infection and its manufacture from presumably infected milk prohibited.

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[a] Bruck, Deut. Med. Woch., vol. 29, 1903, p. 460.

[b] Washburn, Washington Medical Annals, Vol. VII, No. 1, 1908, p. 107.

Buttermilk of course would be fully as dangerous as the cream from which it was derived.

Cheese.—The time required for the ripening of cheese makes the chances of infection from this dairy product certainly very slight.

Butter and cheese from a given source are usually so widely distributed that should an outbreak of typhoid fever in a large city be caused by infection in them it would be very difficult to trace unless the outbreak were very pronounced and other possible factors could be excluded so that attention would be directed to these dairy products.

WAYS IN WHICH THE TYPHOID BACILLUS MAY GET INTO MILK.

At the dairy farm.—The milk supply for the average American city is obtained from a large number of dairy farms, and the liability of cases of typhoid fever developing every year among persons living on these farms can be readily appreciated. The city of Washington, for example, obtains its milk supply from about 1,000 dairy farms. Estimating the average number of persons living at a dairy farm at about 7, and considering the fact that every year, in the United States, about 1 person in every 300 has typhoid fever, some 25 cases per year may be expected to occur on the dairy farms supplying Washington with milk.

When cases exist on the dairy farms, there is, in the majority of instances, much likelihood of the infection being conveyed from the patients to the milk. Frequently the cases are not recognized as typhoid until the second or third week of illness, during which period no precautions are taken. In many instances there are mild cases unattended by a physician and cases following an irregular course, which go through the attack without being recognized. Too frequently when the cases are correctly diagnosed in the comparatively early stages of the disease the disinfection of the patient's stools and urine and the other precautions necessary to prevent the spread of the infection are found to be woefully inefficient.

When the infection is not destroyed as it leaves the body of the patient, there are many ways in which the typhoid bacilli may be carried from a patient on a dairy farm to the milk. Thus, those caring for the sick or handling the soiled bedding or excreta of the patient may convey the infection on their hands or clothing. Persons who have recovered from the symptoms of the disease, but are still discharging the bacilli in their stools or urine may directly contaminate the milk in handling it. Some persons after passing through an attack of typhoid fever continue to discharge the bacilli in their stools or urine for years. One of these "bacillus carriers" working in milk at a dairy farm or dairy may contaminate a can of milk from time to time and be the source of infection for a number of cases.

There have been reported recently several outbreaks of milk-borne typhoid fever traced to infection from bacillus carriers. Albert[a] reports an outbreak in October, 1907, at Cedar Falls, Iowa, in which 13 cases occurred in the three families supplied with the suspected milk. The man who owned the cow and did the milking had had typhoid fever one year previous to the outbreak. He was found to have a slight cystitis and typhoid bacilli were demonstrated in the urine. No source other than the urine of this man could be discovered to account for the infection in the milk.

Lumsden and Woodward[b] report an outbreak in September and October, 1908, in Washington, D. C., in which 54 cases occurred. The cases were among the customers of two different dairymen, both of whom, however, received a part of their milk supply from a certain farm. All the evidence obtained pointed to this farm as the source of the infection in the milk. No history of recent sickness on the farm could be ascertained. A search for bacillus carriers among the persons on the farm was made and in the specimen of feces obtained from one of the milkers—a woman who had had an attack of typhoid fever about eighteen years before the time of the outbreak—typhoid bacilli were found in large numbers. No source other than the feces of this woman could be discovered to account for the infection in the milk.

Scheller,[c] in the course of an investigation of an unusual prevalence of typhoid fever in a section near Königsburg, Germany, directed his attention to the milk supply. He discovered, among those who handled the milk, a woman who was excreting in her stools and urine typhoid bacilli. There appeared to be no doubt that this woman, who had had an attack of typhoid fever seventeen years before, constituted the source of infection for the outbreak. There were on the estate 180 persons; of these, 140 neither consumed nor in any way handled the milk and the excreta of all of them were found to be free from typhoid bacilli. Of the 40, however, who either handled or consumed the milk, 18 were found to be bacillus carriers. Only 4 of the 18 had had an attack of typhoid fever and these 4 had had the attack some years before. Some of the carriers were excreting the typhoid bacilli in the feces, some in the urine, and some in both feces and urine. The large number suggested that they might be temporary or acute carriers. The woman originally discovered to be a carrier was removed from the dairy. The milkers were required to wash their hands in a disinfectant solution before milking. Those who had typhoid bacilluria were treated with urotropin. Within a month after these precautions were taken the stools and urine of all the carriers were again examined bacteriologically and all found negative except those of the original woman, whose excreta still contained typhoid bacilli in large numbers.

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[a] Hyg. Lab. Bull. No. 41, Jan., 1908, p. 19.

[b] Journ. Am. Med. Assn., March 6, 1909, vol. LII, pp. 749-752.

[c] Centblt. f. Bakt., vol. 46, 1908, p. 385.

The Strasbourg School has found, in a number of instances, such temporary or acute carriers among healthy persons living in close association with typhoid fever cases.

The possibility of there being chronic or acute bacillus carriers among those concerned in handling the milk should be kept in mind in the investigation of a suspected milk supply.

It is possible for persons in the early stage of the disease, and even before becoming ill enough to take to bed, to contaminate milk. The spread of infection from cases in the early stage has generally been considered of infrequent occurrence on the ground that the bacilli rarely appear in the urine before the end of the third week of illness and that few if any are discharged in the feces during the first week or two. On the contrary, H. Conradi,[a] who has made extensive studies on the conveyance of typhoid infection in Germany, states that he has reached the conviction that not only is the infection transmitted most often during the earliest stages of the disease, before its true nature has been recognized, but that it also frequently takes place during the incubation period. He bases this opinion on the observation that of 89 cases which he attributed to infection by contact, some 58 per cent of the secondary cases had onset of illness within a week after the onset of illness of the primary cases.

Flies passing from infected excreta to the milk or the milk cans may readily convey the infection.

The excreta of patients thrown into the privy or in the yard or field near by may be carried by drainage, seepage, on the feet of persons, etc., to the well, spring, or stream from which water is used for washing cans, and so be conveyed to the milk.

In country places there are frequent instances where chickens and other fowls have free access to the privy contents and may readily carry infection on their feet to the well or spring or to the dairy-house yard in which milk cans are set. The excreta of patients, carelessly handled, may become dried and carried as dust into exposed milk or, more frequently perhaps, into exposed milk vessels.

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[a] Deut. Med. Woch., Oct. 10, 1907.

Bottles or cans in some way contaminated at the home or dairy in the city and without previous disinfection are again filled with milk at the dairy farm may be the means of conveying infection from the dairy farm back to the city.

At the dairy.—Milk after it reaches the city dairy is exposed again to the danger of becoming contaminated by persons handling it or by flies, dust, etc.

At the average large city dairy there are a number of employees who reside at their own homes. At times some of these persons may come directly from the bedside of a case of unrecognized typhoid fever in the family to the dairy and, as is the case too frequently, without being required to change their clothes or to wash their hands in a germicidal solution, engage in handling the milk.

In some American cities many of the dairies are located in the most unhygienic sections, and frequently cases of typhoid fever are cared for in houses adjoining the dairy or even in the same building. In these instances it is easy to understand how flies may pass from the dejecta of a patient to a can or bottle of milk and so be the means of conveying the infection. Cans or bottles returned from houses in which there are typhoid patients and which have been handled by persons caring for the sick and not disinfected before being refilled may be the means of disseminating the infection in the milk.

From the water used for washing the bottles or cans, etc., at the city dairy, the typhoid bacillus may reach the milk. Considering the immense dilution in which the typhoid bacillus must usually exist in water taken from a large volume, such as a river or lake, for supplying a city, it may be that persons are rarely infected directly by the organism in the water; but the occasional typhoid bacillus in the water, upon being introduced into the milk and there multiplying, may infect persons drinking that milk.

At the grocery.—In the studies of Rosenau, Lumsden, and Kastle[a] on the prevalence of typhoid fever in the District of Columbia there were found a number of instances in which typhoid patients were being cared for in rooms above or to the rear of small grocery stores. In these stores milk was sold in small quantities, often as little as a cent's worth at a time, so that a quart bottle would be divided among several customers. The same hands that nursed the patient purveyed the milk. In such instances not only is there a likelihood of infection being sent out in the milk directly from the store, but these much-handled bottles may do damage when returned to the dairy.

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[a] Hygienic Laboratory Bulletin No. 35, Report on the Origin and Prevalence of Typhoid Fever in the District of Columbia, 1907.

At the home.—Milk after being delivered to the house may become contaminated by the hands of those caring for the sick or by flies, etc., and be the medium of conveyance of infection to other members of the household.

DETERMINATION OF AN OUTBREAK OF TYPHOID FEVER DUE TO INFECTED MILK.

In the epidemiological studies of typhoid fever in a city a card should be kept for each milk dealer and on this card should be noted all cases of typhoid fever in persons who within thirty days previous to onset of illness have used milk supplied by that dealer. Thus, as soon as an unusual number of cases are reported along the route of any dairyman it is apparent on the card and attention may be given at once to the dairy and the farms supplying the dairy with milk.

A number of conditions should be taken into consideration in determining what constitutes an unusual number of cases among the customers of a given dairyman. Of those conditions to be especially considered are the general prevalence of typhoid fever in the community, the amount of milk sold, the method of handling the milk at the dairy, the number of sources from which the milk comes to the dairy, and the way in which milk is served to customers.

Ten or fifteen cases occurring in the course of ten days among the customers of a dairyman who sold 1,000 gallons of milk daily, and who at his dairy mixed the milk received from the various dairy farms supplying him before delivering it to his customers, might not impress an investigator as being an unusual number of cases, especially if typhoid fever was generally quite prevalent in the community and the cases among the dairyman's customers were distributed over a large section of the city. On the other hand, if it were learned that at the dairy the milk was bottled directly from the individual cans as they came from the different farms, and that the 10 or 15 cases had occurred among persons who had been served with milk from one farm which supplied the dairy with 10 or 20 gallons of milk daily, suspicion would fall at once upon the milk.

In the first case, however, the milk might have been equally at fault, the infection having been originally in one can of milk as it came from the farm; but as the milk in this can was mixed with a large volume of other milk in which, due to temperature, lack of time, or other conditions, the infective organisms did not undergo much multiplication, and so were distributed in high dilution in the milk, even as they may be at times in cases of water infection.

In order to properly charge to each dairyman the cases having used milk supplied by him it is necessary to take into consideration not only the source of the milk used regularly by the patient during the thirty days previous to onset of illness but also of that used occasionally. Frequently it will be found that a family receiving its regular milk supply from a certain dairy will on occasions, when the regular supply is not sufficient for the needs of the day, obtain milk from some other dairy, directly or through the grocery store. The milk obtained on one of these occasions may be infected and so responsible for the case. The source of milk used at places other than the regular one for taking meals also should be ascertained if possible.

Cases resulting from infection in the milk are by no means confined to persons who use milk as a beverage. The cream or milk used on cereals, fruits, or even in coffee may convey the infection. In the summer of 1906 in Washington there were six cases in one family of eight persons, all of which were attributed quite definitely to infected milk. None of the members of this family drank milk, but they all used cream on fruits and cereals. Of course the chances of contracting the infection from milk is greater among persons who use milk freely.

George Newman[a] sums up the characteristics of milk-borne epidemics as follows:

(*a*) There is a special incidence of disease upon the track of the implicated milk supply. It is localized to such area.

(*b*) Better-class houses and persons generally suffer most.

(*c*) Milk drinkers are chiefly affected and they suffer most who are large consumers of raw milk.

(*d*) Women and children suffer most, and frequently adults suffer proportionately more than children.

(*e*) Incubation periods are shortened.

(*f*) There is a sudden onset and rapid decline.

(*g*) Multiple cases in one house occur simultaneously.

(*h*) Clinically the attacks of the disease are often mild. Contact infectivity is reduced and the mortality rate is lower than usual.

In the different outbreaks due to infected milk it is interesting to note how greatly the proportion of persons affected among the users of the milk varies. In some outbreaks the proportion is as great as 25 per cent; for instance, in the epidemic at Palo Alto, Cal., in 1903, which was traced to infected milk by Fish, Mosher, and Snow. Of the 900 persons who used milk from the infected supply, 232 had typhoid fever. In other outbreaks the proportion is as low as 1 or 2 per cent. Several conditions no doubt influence the proportion of persons affected, the most important of which probably is the amount of infection in the milk. In the Palo Alto epidemic it was determined that the milk became infected through the water used for washing the cans and also at times for diluting the milk. This water was obtained from a creek which received the drainage from several houses in which there were patients with typhoid fever. The water of the creek for two or three weeks must have been quite heavily charged with typhoid bacilli, so that probably the majority of the milk cans washed in this water received some of the organisms.

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[a] George Newman, Bacteriology and the Public Health, 1904.

It is easy to understand how a milk supply thus almost if not quite continuously infected for several weeks may cause the infection of a large proportion of the persons who use that milk; but when the infection is introduced into the milk at irregular intervals for a like period, as would be expected when the infection is conveyed on the hands or clothing of persons or by flies, etc., a very small proportion of the consumers of the milk may become infected.

The susceptibility of the people supplied with infected milk, of course, would affect the proportion. In a community where typhoid fever had been prevalent for years, and in which there would be a number of persons rendered relatively immune by previous infection, we would expect less susceptibility than in a community where the disease had never prevailed.

That it takes susceptibility plus exposure to infection for the disease to occur was strongly suggested by an instance in the course of a milk outbreak in the District of Columbia in the fall of 1906. In a children's home having about 100 inmates 7 children came down with typhoid fever within a period of two or three days. The way in which the milk was delivered to and served at the institution made it practically impossible for the 7 children affected to have drunk milk from any one can, or one day's delivery, from which at least 75 per cent of the children did not drink. Thus of 75 children almost certainly drinking infected milk only 7 had the disease. It is conceivable that in such an instance the typhoid bacilli in the can or cans of infected milk either were very few in number or that they were not uniformly distributed through the bulk of the milk, so that only one or two of the children drinking from a 5-gallon can of milk actually received any of the bacilli; but it seems much more reasonable to conclude that all of the 75 children received some of the bacilli and the escape of the majority was due entirely to lack of susceptibility at the time the organisms were ingested.

It seems quite probable that different strains of the typhoid bacillus vary markedly in their infectiveness. The writer has become impressed with this view by observing in the course of his studies of typhoid fever in the District of Columbia frequent instances in which there are one or more cases of typhoid fever in a household in a most unhygienic and crowded neighborhood, many persons having free association with the patients, the excreta of the patients being handled with the grossest carelessness, flies swarming over the excreta as well as over the food for the sick and well, and yet under these apparently very favorable conditions for the spread of typhoid fever infection not a single secondary case develops among other persons in the household, or even in the neighborhood. In other instances, one or more cases are being cared for in a household in good sanitary surroundings, ordinary care as to cleanliness, disposal of patients' excreta, personal contact with patients, etc., being exercised, and yet two or three or more secondary cases develop among other persons in the house or in houses near by.

Of course in making a comparison of such instances it can not be stated how much the results are affected by individual susceptibility or by the operation of some as yet unknown factor or factors in the conveyance of or in the establishment of susceptibility (perhaps specific) to typhoid-fever infection.

It may be readily understood how strains of the organism of a low degree of infectiveness (and of virulence) getting into milk, there undergoing tremendous multiplication and so being distributed in large doses, may cause outbreaks of typhoid fever. This view of low infectiveness and virulence of the organism being offset by large dosage is supported by some of the features observed in a number of the reported milk-borne outbreaks. For instance, short period of incubation, sudden onset and rapid decline of attack, reduced contact infectivity and low fatality rate.

It is theoretically possible, however, that these features are due to large dosage alone. Thus, a large number of virulent organisms upon being introduced suddenly into relatively highly resistant tissues, a pronounced reaction occurs (sudden onset of definite symptoms) with resulting formation of relatively large amount of antibodies (rapid decline of attack, reduced contact infectivity, etc.).

The establishment of milk as the causative factor in an outbreak of typhoid fever is based on the following points:

(*a*) A sudden and marked increase in the number of cases along the route of some dairyman, without a corresponding increase in the number of cases among persons living in the same sections of the city but supplied with milk from other sources. In a town supplied largely or entirely by one dairyman a sudden increase in the number of cases would not implicate the milk unless other facts pointed to it and other factors could be excluded, but in large cities, where the people of practically every square are supplied with milk by two or more dairymen, an increase in the number of cases distinctly on the route of a given dairyman is quite easily determined. This fact alone is evidence that the milk is responsible, and if an investigation reveals that at a time corresponding to the period in which the group of cases along the dairyman's route became infected there was at the dairy or one of the dairy farms a patient with typhoid fever whose discharges could readily have reached the milk, the chain of evidence is sufficiently strong to justify the assumption that the outbreak was due to the milk supplied by this dairyman, especially if the cases can not positively be proven to have been due to some other factor.

(*b*) The demonstration of the typhoid bacillus in the suspected milk. When this is done, the chain of evidence is, of course, complete. But frequently it can not be done, because in the period of usually three or four weeks—covering the incubation period, diagnosis, and report of the cases—elapsing between the time of infection of the cases and the recognition of the outbreak, the typhoid bacillus has disappeared from the milk.

If cases of typhoid fever are not discovered to account for the infection of an implicated milk supply, it is well to examine bacteriologically the stools and urine of all persons who handle the milk at the farms and the dairy. In this way the source of the infection may be found in the discharges of some person who has the disease in an ambulant and unrecognized form (temporary bacillus carrier) or of some one who has been carrying the infection for months or even years (chronic bacillus carrier).

Besides the large groups of cases of typhoid fever caused by infected milk, there must be in large cities frequently single cases or small groups of cases which are due to infection in the milk and yet can not be traced to that source. In a community where factors other than milk were operating to cause a rather extensive prevalence of typhoid fever, 5 or 6 cases occurring within a few days among the customers of a dairyman supplying several hundred families with milk would direct some suspicion toward that milk supply, but if this small group of cases should not be followed by an unusually large number of cases on the route of this dairyman and no typhoid cases were found on the dairy farm or at the dairy, these 5 or 6 cases would be placed by the investigator among those due to causes undetermined or to causes other than milk. In many such instances, however, these groups of cases are doubtless due to infection introduced in one of the many possible ways—hands, clothes, flies, water for washing cans, etc. into a part of the dairyman's output of milk for perhaps only one day.

In cities having milk supplied by a number of dairymen, if several of these small groups of cases among customers of different dairymen occur at about the same time, a list of the farms supplying each of the suspected dairies should be studied, and if it is found that two or more of these dairies receive milk from any one farm, an investigation should be made of that farm, and in this way the source of the infection for the several groups of cases may be determined.

MEASURES TO PREVENT THE DISSEMINATION OF THE INFECTION OF TYPHOID FEVER IN MILK.

(*a*) *The prevention of the introduction of infection into milk.*—This at once suggests itself as the proper measure; but the difficulty of carrying it out practically becomes evident when we consider the number of farms from which the milk supply of the average American city is obtained, the liability of cases of typhoid fever occurring on these farms, and the numerous ways in which the infection may be conveyed from the patient to the milk. New York City's milk supply, according to Darlington, is derived from 35,000 farms, and shipped from 700 creameries, located in 6 States. It is easy to appreciate how difficult and expensive it would be to keep up a sufficiently thorough supervision of the multiple sources of that city's milk supply. It is practicable to accomplish much toward the prevention of the infection getting into the milk after the milk is delivered to the city. The following requirements are suggested:

1. Location of the dairies in good surroundings.

2. The prevention of the handling of the milk by persons who are in contact with typhoid fever patients or who themselves are liable to be discharging typhoid bacilli in their excreta. It does not seem unreasonable to require the owner of a store in which milk is sold and in which there is a patient with typhoid fever to either remove the patient to a hospital or some other house or to close up the business until the danger from that patient is passed.

3. Exclusion of flies and other insects so far as possible, by screening, etc.

4. Sterilization of bottles and cans returned from houses before being again filled with milk, or the use of paper bottles which would not need to be returned.

5. The sealing of the bottles or cans of milk so that they may not be infected in the course of delivery.

(*b*) *The destruction of infection in milk.*—This at the present time seems to be the cheapest and the most practicable method to prevent the spread of typhoid infection in the milk supply of cities. In exceptional instances when a dairy receives its supply of milk from only one or two farms over which a thorough supervision may be exercised, efforts to prevent the infection reaching the milk may be attempted. But for the general supply of cities officially supervised pasteurization of the milk is the best measure. Supplement this with an intelligent supervision over the depots and stores where milk is sold and milk as a causative factor of typhoid fever in cities would be removed.

In other words, pasteurization appears at the present time to be the only practical solution of the milk problem. All objections to the proper pasteurization of milk seem to be entirely theoretical or such as may be readily overcome.

Of the theoretical objections, one frequently advanced in written or spoken arguments and placarded at model dairy farm exhibits is, "Pure milk is better than purified milk." That may be true; but how can pure milk be obtained in sufficient quantity to supply our larger cities and at what cost?

Milk to be desirably clean must be obtained from especially well-equipped dairy farms and handled entirely by highly skilled and highly conscientious or closely guarded persons. The cost of installing such equipment and the employment of such a class of labor would have to be met by a decided increase in the price of milk, while pasteurization—certainly if done on a large scale—should not increase the price of milk more than a small fraction of a cent on the quart.

Another objection to the pasteurization of milk is that the heat does not remove the objectionable bacteria, but simply kills them, so that the consumer gets the dead bacteria anyhow. That is true; but does it not appear safer to ingest these dead bacteria than to take into the alimentary canal the same bacteria living, which may continue to multiply and generate an increasing amount of products harmful to the human organism? No reasonable advocate of pasteurization can hold that grossly dirty milk should be used as a food, either pasteurized or unpasteurized, the aim being to get milk as pure as practicable and then purify it to a point of safety. Milk containing only 10 bacteria to the cubic centimeter would not be safe if some of those bacteria were typhoid bacilli.

Another seemingly entirely theoretical objection to pasteurization is that the heat changes the milk in some way so that it induces certain diseases, such as scurvy and rickets, or lowers the resistance of persons using it so that they are more liable to certain infections, particularly intestinal diseases. The vast bulk of reliable evidence so far recorded on this subject indicates that this objection is not supported by facts, while there is constantly accumulating indisputable evidence that there is much sickness caused by organisms in raw milk, which organisms would be destroyed by pasteurization.

A removable objection to the pasteurization of milk is that the heat destroys the lactic acid producing organisms which cause the "natural souring" of milk, and leaves organisms which produce other kinds of fermentation ("putrefaction") to flourish. Should this objection prove, by further study, to be valid, it could be met readily by adding to the milk after it is pasteurized some pure culture of lactic acid forming organisms.

It seems that in pasteurization we have a practical, unobjectionable, immediately needed remedy, while in the necessary measures to obtain a pure milk supply for our larger cities we have but a hope for the future.

4. FREQUENCY OF TUBERCLE BACILLI IN THE MARKET MILK OF WASHINGTON, D. C.

THE FREQUENCY OF TUBERCLE BACILLI IN THE MARKET MILK OF THE CITY OF WASHINGTON, D. C.

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INTRODUCTION.

Numerous investigators in recent years have shown the infectiousness of milk containing tubercle bacilli for animals. Whether the milk from animals with tuberculosis but with healthy udders contains tubercle bacilli is not definitely settled. Many prominent scientists seem to have shown that at times the milk from such animals does contain tubercle bacilli virulent for laboratory animals, but in the view of recent work there may be some doubt as to whether the bacilli really passed through the udder but gained access to the milk from contamination with feces containing tubercle bacilli.

Schroeder and Cotton [a] have recently shown that cows so slightly affected with tuberculosis as only to be discoverable by the tuberculin reaction pass virulent bacilli in their feces. Many believe that milk from a tuberculous cow with unaffected udder is free from infection and becomes infected from the feces of the animal or its environment. This observation is of the very greatest importance, and if confirmed shows, more than ever, that the greatest care is necessary in guarding milk from contamination from the time it is drawn until it is consumed.

The milk supply of many of the cities of Europe and England has been examined for tubercle bacilli. Most observers have used the animal test; they have injected various amounts, either centrifugalized or not, into guinea pigs or rabbits. The percentage of samples showing tubercle bacilli has varied between very wide limits, no doubt dependent upon the difference in the number of tuberculous cows in the herds supplying milk to the different cities and on differences in technic. Some observers have found that when a number of animals are inoculated with the same samples of milk only one, perhaps, will develop tuberculosis. Some centrifugalized the milk and gave sediment alone, while others gave sediment and cream.

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[a] Schroeder, C. C. and Cotton, W. E.: Bull. of the Bureau of Animal Industry, 1907.

I will not enter into the question whether the tubercle bacilli found in milk are virulent for man, but give my results solely as to whether the market milk of the city of Washington contains tubercle bacilli virulent for guinea pigs. For myself I object most strenuously to using milk containing tubercle bacilli virulent for laboratory animals and prefer to leave the question as to their pathogenicity for man to be discussed by others.

Before presenting the results obtained by me with the market milk of the city of Washington it will be interesting to refer briefly to results obtained elsewhere by others.

REVIEW OF LITERATURE.

Bang, B. Deut. Zeit. f. Thiermed. XI, 1884, p. 45.

Injected apparently normal milk from the sound quarter of an udder another part of which was diseased, into the belly wall of two rabbits, which developed inoculation tuberculosis and died after 2⅓ and 3⅓ months, respectively. This was repeated later with two more specimens of milk, with the same result. He also demonstrated that the milk of tuberculous cows without demonstrable udder lesions, could contain tubercle bacilli.

Stein, G. Experimentelle Beitrage zur Infektion der Milch perlsuchtiger Kuhe. Inaug. Dissert., Berlin, 1884.

Intraperitoneal inoculation of guinea pigs with raw milk of tuberculous cows. Ten negative and four positive results. In two of the latter tubercle bacilli were demonstrated, and two negative. Some of the cows had tuberculosis of the udder.

Hirschberger, K. Experimentelle Beiträge zur Infectiosität der Milch tuberculöser Kühe. Deut. Arch. f. klin. Med., XLIV, 1889, p. 400.

Twenty specimens of milk from tuberculous cows injected into the peritoneum of guinea pigs. None of the animals inoculated died of septic peritonitis. Eleven of the specimens proved to contain tubercle bacilli. (Other acid-fast organisms, of course, were not differentiated.) By microscopic examination only one of the specimens of milk was shown to contain tubercle bacilli. Tubercle bacilli occurred not only in milk from tuberculous udders, but also where the udders were sound, and where the cow was but slightly affected with tuberculosis.

Gebhardt, F. Experimentelle Untersuchungen ueber den Einfluss der Verdünnung auf die Wirksamkeit des tuberkulösen Giftes. Virch. Arch., CIX, 1890, p. 127.

First series (2.5 cubic centimeters of milk or dilutions injected into guinea pigs intraperitoneally; milk from a tuberculous udder): Undiluted milk and 1 to 20, positive result; 1 to 40 to 1 to 100, negative.

Second series (2 cubic centimeters fluid injected intraperitoneally): Undiluted milk, positive; 1 to 50 to 1 to 200, negative.

Third series (1 cubic centimeter subcutaneously): Undiluted milk and 1 to 50, positive; 1 to 100 to 1 to 1,000, negative.

These results show the effect of dilution of infected milk by uninfected milk, as it will be seen that dilutions of greater than 1 to 50 failed to produce tuberculosis in the inoculated animals.

In an examination of market milk from ten different sources in Munich, 2 cubic centimeters were injected into the peritoneum of guinea pigs with negative results in all cases.

Ernst, H. C. How far may a cow be tuberculous before her milk becomes dangerous as an article of food? Amer. Jour. Med. Sci., XCVIII, 1890, p. 439.

1. Microscopic examination of cover-glass preparations made from milk of tuberculous cows without udder tuberculosis. Various parts of milk and cream examined:

Specimens examined	114
Specimens containing tubercle bacilli	17
Per cent	31.5
Cows examined	36
Cows having tubercle bacilli in milk	10
Per cent	27.7

2. Inoculation of rabbits (method not stated) with similar milk:

Rabbits surviving first few days, etc	49
Rabbits becoming tuberculous	5
Per cent	10.2
Cows used	13
Cows with milk shown tuberculous	3
Per cent	23

3. Inoculation of guinea pigs (method not stated) with similar milk:

Guinea pigs after necessary exclusions	54
Guinea pigs becoming tuberculous	12
Per cent	22
Per cent (author says)	28.57
Cows used	14
Cows giving tuberculous milk	6
Per cent	42.8

4. Feeding calves with similar milk, 5 out of 12 (41.66 per cent) became tuberculous.

5. Feeding pigs with similar milk, 2 out of 5 (40 per cent) became tuberculous.

McFadyean & Woodhead. On the transmission of tuberculosis, etc. Internat. Cong. Hyg. and Demog., 1891, sec. 2, p. 197.

Inoculations with tuberculous udder juice and milk from tuberculous udders 70 per cent were positive (14 of 19). Inoculations with nontuberculous udders and milk from tuberculous cows (udders not affected), 16 per cent were positive (2 of 13).

Bang, B. Experimentelle Untersuchungen ueber tuberculose Milch. Deut. Zeit. f. Thiermed. XVII, 1891, S. 1.

Examined the milk of 28 cows having advanced tuberculosis, but no udder involvement. Rabbits injected, with 1 or 2 cubic centimeters intraperitoneally. The milk of two of these cows was shown to contain virulent tubercle bacilli.

Fiorentini, A. Giornale della R. Soc. d'igiene. 1892, p. 198. (Ref. in Baumgartens Jahresb., 1892, p. 698.)

Injected the milk of tuberculous cows into the peritoneum of guinea pigs, with positive results (tuberculosis) in three cases. In two of these there was udder tuberculosis.

Friis, St. Beitrag zur Beleuchtung der Frage ueber die Ansteckungsgefahr der Handelsmilch mit bezug auf die Tuberkulose. Deut. Zeit. f. Thiermed., Bd. XIX, 1893, p. 115.

Samples of mixed milk from 46 establishments in and about Copenhagen were examined. Experiments from May to October. Eighteen samples must be excluded from consideration on account of the early death of the inoculated animals. Of the remaining 28 specimens, 4 were found to contain tubercle bacilli (14.3 per cent). One of the positive specimens was from a herd of 30 cows, only 1 of which was suspected of having tuberculosis, showing the danger of diluted tuberculous milk. The other milk in which the tubercle bacilli was found was from dairies having one or more tuberculous cows.

Friis, St. Fortgesetzte Untersuchungen u. s. w. Deut. Zeit. f. Thiermed. Bd. XX. 1894, p. 195.

In a former paper, *q. v.*, the author has considered town milk, from Copenhagen. He now investigates country milk, taking the specimens at the railroad station upon the arrival of the milk. Experiments from January to May; the former examinations were at a later time in the year.

Five cubic centimeters each of 40 specimens were injected intraperitoneally into rabbits. Seven specimens excluded by early death of the animals. No tubercle bacilli were demonstrated in any of the remaining 33 specimens, although in one instance the findings were extremely suspicious. Consequently the country milk is regarded as being much freer from tubercular infection than the town milk. Also a much smaller percentage of animals died of peritonitis when injected with the country milk.

Schroeder, E. C. Further experimental observations on the presence of tubercle bacilli in the milk of cows. Bulletin No. 7, B. A. I., Agric. Dept. 1894, p. 75.

1. Samples of mixed milk from dairies. Forty cubic centimeters of milk centrifuged, 5 cubic centimeters of sediment layer injected into the peritoneum of guinea pigs. Other pigs inoculated with 5 cubic centimeters of the whole milk. Of 19 specimens, 1 apparently contained tubercle bacilli as the animal receiving the whole milk died of tuberculosis. Its companion getting the centrifuged sediment remained normal.

2. Samples of milk from tuberculous cows diagnosed clinically or by tuberculin. Milk of 12 such cows injected into guinea pigs. Only 1 showed tubercle bacilli.

3. Repeated injections into the same guinea pig of milk from the same tuberculous cow not having udder tuberculosis. Four such cows used, from 2 to 7 pigs receiving several injections of the milk of the same cow. None of the pigs became tuberculous.

The author concludes that careful inspection of all dairy herds, which has for its object the detection and removal of all advanced cases of tuberculosis, and especially of cows with diseased udders, would probably exclude the sale of most infected milk.

Ernst, H. C. Article on The Infectiousness of Milk, Boston, 1895. Pub. by Soc. for Promoting Agriculture.

Modifies the statements of results made in a former article which was published before the completion of the experiments.

A. Milk from cows having tuberculosis, but healthy udders.

1. Cover-glass examinations. Thirty-six cows examined; tubercle bacilli in milk of 12 (33.33 per cent).

2. Subcutaneous inoculation of guinea pigs. Eighty-eight guinea pigs inoculated; 12 became tuberculous. Fifteen cows examined; tubercle bacilli in milk or cream of 6 (40 per cent).

3. Subcutaneous inoculation of rabbits. Ninety rabbits inoculated; 6 became tuberculous. Nineteen cows examined; tubercle bacilli in milk of 4 (21 per cent).

4. Feeding rabbits, details not given. Forty-eight rabbits fed; 2 became tuberculous. Five cows examined; tubercle bacilli in milk of 1 (20 per cent).

5. Feeding pigs. Ten pigs fed; 5 became tuberculous.

6. Feeding calves. Twenty-one calves fed; 8 became tuberculous.

B. Milk at random from Boston supply.

1. Cover-glass examination; 1 specimen out of 33 contained tubercle bacilli.

2. Inoculation of rabbits. Three out of 25 rabbits became tuberculous. (From the tables it appears that 3 of 13 specimens contained tubercle bacilli, although this is not stated in the text.)

C. Of 19 calves born of tuberculous cows, and autopsied within six days of birth, no evidence of tuberculosis was found.

Obermüller, Kuno. Ueber Tuberkelbacillenbefunde in der Marktmilch., Hyg. Rundsch., V, 1895, No. 19, p. 877.

At first injected the milk without centrifuging. Some, at least, of the specimens had been freed from the slime layer in the creamery. Of 40 guinea pigs inoculated, 3 died of peritoneal tuberculosis. Eight, however, had died within a few hours of inoculation. Later he improved his technic by first centrifuging the milk and then injecting a mixture of the cream and sediment layers. By this method 38 per cent of all the animals injected became tuberculous. (Although the author does not specifically state it, it appears from the tables that of the 19 specimens the animals injected with which remained alive long enough to determine the presence of tuberculosis, 9 contained tubercle bacilli.

Buege, A. Ueber die Untersuchung der Milch auf Tuberkelbacillen. Inaug. Dissert., Halle, 1896.

Nine specimens of Halle market milk were injected into 17 guinea pigs intraperitoneally. Three specimens were excluded on account of the early death of the animals. In 2 of the remaining 6 specimens tubercle bacilli were demonstrated by the findings in the animals after death. He injected 5 cubic centimeters of a mixture of cream and sediment from the centrifuged 40 cubic centimeters sample used in each case.

Delepine, S. Jour. Comp. Path. and Ther., vol. 10, pp. 150, 189.

By microscopic examination found tubercle bacilli in 4 out of some 40 specimens of unmixed milk. By the inoculation method, 20 to 25 per cent of these milks were found to be tuberculous. He prefers the subcutaneous method of inoculation to the intraperitoneal, as being more delicate.

Hope, W. E. Report of the Medical Officer of Health, Liverpool, 1897, on tuberculosis as affecting the milk supply of the city.

Two hundred and twenty-eight samples of milk from town dairies were examined and 12, or 5.2 per cent, were found to contain tubercle bacilli. Sixty-seven samples from country dairies showed 9, or 13.4 per cent, with tubercle bacilli. The work was done by Boyce, Delepine, Hamilton, and Woodhead. Animal inoculations, intraperitoneal or subcutaneous, of plain milk or of the sediment after centrifuging.

Massone, A. Annali d'igiene sperimentale, 1897, p. 239. (Ref. in Hyg. Rundsch., VIII, 1897, p. 605.)

Examined a large series of samples of Genoa market milk for the presence of the tubercle bacillus. Centrifuged 70 to 80 cubic centimeters of the mixed milk for 15 minutes, and then injected 5 to 6 cubic centimeters of a mixture of the cream and sediment into the peritoneum of guinea pigs. In 9 per cent of the cases tubercle bacilli were demonstrated in the milk by these means.

Ott. Ein weiterer Beitrag zur Milchhygiene. Zeit. f. Fleisch und Milchhygiene, 1897, VIII, p. 69.

Examined specimens of mixed market milk for the presence of tubercle bacilli. By staining specimens of the milk, treated by a special process, he demonstrated tubercle bacilli in 5 out of 43 specimens. = 11.6 per cent. Guinea pigs were then inoculated intraperitoneally with 5 cubic centimeters of a mixture of cream and sediment of centrifuged milk, obtained from the dealers who had furnished the tuberculous specimens.

Specimen.	Tubercle bacilli microscopically.	Inoculation result.
I	A few	Pig 1 died in 23 days; tuberculous. Tubercle bacilli found. Pig 2 killed in 30 days; tuberculous. Tubercle bacilli found.
II	+ first examination; − second.	Both pigs normal after 5 weeks.
III	5 per field	Pig 1 died in 28 days; tuberculous. Tubercle bacilli found. Pig 2 died in 35 days; tuberculous. Tubercle bacilli found.
IV	Few	Both killed in 6 weeks. First normal; second tuberculous. Tubercle bacilli found.
V	do	Pig 1 died in 40 days; tuberculous. Tubercle bacilli not mentioned. Pig 2 killed in 40 days; tuberculous. Tubercle bacilli not mentioned.

In another series, 30 specimens of market milk were injected intraperitoneally into 30 guinea pigs, 5 cubic centimeters each. Six animals died of intercurrent diseases, only 2, however, too early for the development of tuberculosis.

Four died of tuberculosis, but it was subsequently found that 2 of them had received milk from the same dealer.

To sum up (after making the necessary exclusions), of 27 (author says 28) specimens, 3 contained virulent tubercle bacilli, (11.1 per cent) (author says 10.7 per cent).

Delepine, S. Brit. Med. Jour., 1898, vol. 2, p. 918.

In a popular lecture, gives the following results with milks collected by health officers of Liverpool, Manchester, and elsewhere:

(*a*) Seven specimens unmixed milk from cows showing no evidence of tuberculosis. Tubercle bacilli in none of the specimens.

(*b*) Twenty-two specimens unmixed milk from cows showing distinct evidence of tuberculosis and in 6 cases udder involvement. Tubercle bacilli in 27.24 per cent.

(*c*) Fifty-four specimens mixed town milk. Tubercle bacilli in 5.55 per cent.

(*d*) One hundred and twenty-five specimens country farm milk. Tubercle bacilli in 17.6 per cent.

The presence of tubercle bacilli was determined by inoculation of guinea pigs and their post-mortem examination.

Petri. Zum Nachweis der Tuberkelbacilli in Butter und Milch. Arb. a. d. kais. Ges.-Amt., XIV, 1898, p. 1.

Milk specimens taken from various places in Berlin. Centrifuged in 150 cubic centimeter flasks. Three cubic centimeters each of cream, skim milk, and sediment injected into 4 guinea pigs (12 animals for each specimen). Later, on account of the lack of animals, 5 cubic centimeters from each specimen were inoculated into each of 4 guinea pigs.

Sixty-four specimens were examined. Tubercle bacilli were demonstrated in nine (14 per cent). Tubercle bacilli-like rods, not true tubercle bacilli in 4 specimens (6.3 per cent).

It appears that 200 out of the 478 animals died, mostly of peritonitis within the first three weeks, thus eliminating 7 specimens from consideration, and leaving 57 on which to base a percentage of incidence. As 9 of these contained tubercle bacilli, the corrected percentage would be 17.5.

The importance of using a large number of animals for each specimen is shown by the fact that in only 3 of the 9 positive specimens did more than 1 animal become tuberculous. In these 3 cases there were 2.

Ascher. Untersuchungungen von Butter und Milch auf Tuberkelbacillen. Zeit. f. Hyg., Bd. 32, 1899, S. 329.

Injected 17 specimens of Koningsberg milk into guinea pigs intraperitoneally. One of the animals became tuberculous. The milk was partly centrifuged, and the cream and sediment injected, and partly uncentrifuged. No other acid-fast bacilli found.

The first streams from the milking were used, which may account for the lower percentage of infected specimens detected by him than by Rabinowitsch, who used the last part of the milking. The comparison of results with these different portions of the milking may throw light upon the source of infection of the milk, whether from feces or from the milk glands.

Jaeger. Ueber die Möglichkeit tuberkulöser Infektion des Lymph-systems durch Milch und Milchproducte. Hyg. Rundsch, 1899, IX, p. 801.

Examined the milk supplied to a large hospital in Königsberg. The dairy was in good condition and frequently inspected, but the cows were not tested with tuberculin.

Six guinea pigs were injected with the milk intraperitoneally. Two died of sepsis, 2 remained normal, and 2 developed tuberculosis.

One hundred specimens were examined by the coverglass method for the tubercle bacillus, which was demonstrated in 7 specimens.

Kanthack, A. A., and Sladen, E. S. St. B. Influence of the Milk Supply on the Spread of Tuberculosis. Lancet, 1899, vol. I, p. 74.

Examined the milk supply of the various colleges in Cambridge for the presence of the tubercle bacillus. Milk from 16 dairies was examined, 3 specimens from each. Two guinea pigs were injected subcutaneously with each specimen, one from the cream layer and the other from the sediment, after centrifuging 10 cubic centimeters of the milk for minutes; guinea pigs examined after death from disease or killed, the characteristic histological tubercle being deemed necessary for the diagnosis of tuberculosis. Of 33 animals suspected of being tuberculous 10 were found by microscopical examination to be free from the disease, while of 23 having typical histological tubercular lesions, 16 showed the presence of the bacillus.

Results: Of 16 daries examined, 9 furnished tubercular milk. Of 90 guinea pigs inoculated, 23 died from tuberculosis (25.55 per cent). It is interesting to note that 13 of these were inoculated with the cream layer, while only 10 received the sediment.

Macfadyen, Allan. Lancet, 1899, vol. II, p. 849.

In a report of work done at the Jenner Institute for the Hackney vestry, it appears that of 100 specimens submitted for examination 23 had to be excluded from the results because of the premature death of the test animals. Of the remaining 77 specimens, 17, or 22 per cent, were found to be infected with virulent tubercle bacilli. The milk was centrifuged 30 minutes, the cream removed and the milk recentrifuged for 30 minutes. The sediment was then used for inoculating guinea pigs.

Ostertag. Zeit. f Fleisch- und Milchhygiene, IX, No. 12, 1899, p. 221.

Examined the milk of some 50 cows which had no clinical evidence of tuberculosis, but had reacted to tuberculin. Milk received with complete precautions into liter flasks and immediately cooled. The cream rose during transportation and was pipetted off, and to it was added enough of the milk to make 80 cubic centimeters. This mixture was then centrifuged, and a mixture of cream, skim milk, and sediment injected into guinea pigs. Three or four animals were injected with 10 cubic centimeters of each specimen. Each specimen was also examined microscopically for the presence of tubercle bacilli and the remainder was fed to guinea pigs.

Tubercle bacilli were not found in any specimen of the milk by microscopic examination. No pseudo-tubercle bacilli were found. Only 1 animal contracted tuberculosis out of all those injected, representing 1 specimen of 49. The other 3 animals receiving this same milk remained healthy and proved normal on section. The authors, for reasons which they give, do not regard this one case of tubercular infection as being due to the milk. They conclude that there were no tubercle bacilli in any of the 49 specimens. Fourteen specimens of the mixed milk from this herd were then examined. Only 11 remained for consideration. One of the injected guinea pigs was found tuberculous on being killed after seventy-one days, but the lesions were slight and the animal had lost only 20 grams. None of the fed animals became tuberculous.

Rabinowitsch, Lydia, and Kempner, Walter. Zeit. Hyg. XXXI, 1899, p. 137.

Recalls the results of earlier experiments of Rabinowitsch, in which of 25 samples of Berlin milk examined (1897), 7 (28 per cent) contained tubercle bacilli. The milk was centrifuged and a mixture of the cream and sediment layers injected into the peritoneum of guinea pigs.

The present article deals with an examination of the milk of cows reacting to tuberculin. Of 14 such cows, 10, or 71.4 per cent, gave milk containing tubercle bacilli. The condition of these cows is here detailed: Only 1 had pronounced udder tuberculosis. Another had udder tuberculosis demonstrable only histologically. Three cows with advanced generalized tuberculosis gave histologically the picture of chronic interstitial inflammation of the udder. One cow had low grade tuberculosis. One had râles on one examination, but none on the next two. Two cows had no symptom of tuberculosis. Another showed symptoms of beginning tuberculosis only on the second and third examinations.

This demonstrates that in beginning tuberculosis without discoverable udder disease, and in latent tuberculosis demonstrable only by the tuberculin reaction, the tubercle bacilli may be present in the milk. They believe that repeated examination would have shown tubercle bacilli in the milk of more of these cows.

Boyce. (Results given by Annett, Lancet, 1900, p. 160.)

He examined the market milk of Liverpool, England; his results are given in the following table:

	Per cent tuberculous.
Year 1898:	
Town milk (75 specimens)	6.6
Country milk (28 specimens)	17.8
Year 1899:	
Town milk (75 specimens)	6.6
Country milk (63 specimens)	17.4

The superiority of the town milk is attributed to the inspections conducted in town.

Rabinowitsch, Lydia. Deut. med. Woch., XXVI, 1900, p. 416.

Repeatedly examined the milk of eight Berlin dairies. This milk was designed especially for the use of children, was not sterilized, and sold for 35 to 60 pfennig per liter. In three of these dairies the cows were rigidly tuberculin tested. No tubercle bacilli were ever found in this milk. In the other five the cows were subjected to clinical oversight by veterinarians, but the tuberculin test was employed only now and then upon suspicious animals. In three of these five dairies the milk was found to contain tubercle bacilli. The percentage of specimens containing tubercle bacilli is not stated.

Klein. Zur Kenntnis der Verbreitung des Bacillus tuberculosis and pseudotuberculosis in der Milch sowie der Biologie des Bacillus tuberculosis. Centralbl. f. Bakt., 1900, 1. Abt., v. 28, Orig., p. 111.

Klein examined 100 samples of milk from various country farms in the vicinity of London. The samples were placed in conical glasses and allowed to sediment. Smears were made from the sediment and examined microscopically for tubercle bacilli; guinea pigs were also inoculated subcutaneously and intraperitoneally with the sediment.

Klein's results were: Eight guinea pigs died acutely, 7 showed positive tuberculosis, while 42 gave negative results at autopsy. The remainder showed staphylococcic and Streptococcic infection.

Tonzig. Ueber den Antiel, den die Milch an der Verbreitung der Tuberkulose nimmt, mit besonderen Untersuchungen ueber die Milch des Paduaner Marktes. Arch. f. Hyg., 1900, v. 41.

This author examined the market milk of Padua. Forty-six samples were centrifugalized and the cream and sediment injected intraperitoneally into 103 guinea pigs. Nine died within forty-eight hours, and none of the remainder when they were killed showed tuberculosis. Tonzig is of the opinion that the danger of infection with tubercle bacilli in mixed milk is only slight.

The tubercle bacillus in milk. Swithinbank & Newman's Bacteriology of Milk, 1903, p. 213.

During 1901, 310 samples of milk were taken at the Manchester (England) railway station from the milk cans representing 272 farms. One hundred and seventy-two of these farms were in Cheshire, and 18 of them (10.46 per cent) supplied milk found to contain the tubercle bacillus; 65 were in Derbyshire, and 6 (9.23 per cent) supplied milk infected with tubercle bacilli; 25 in Staffordshire, of which 2 (8 per cent) supplied tuberculous milk.

Thus the milk sent by rail to Manchester from 272 farms, and examined by Professor Delepine, was tuberculous from 26 of the farms (9.5 per cent). (See Report Health City of Manchester, 1901, p. 238.)

Collingridge. Tubercle bacilli in milk. (Editorial Abstract in Brit. M. J., 1907, v. 1, p. 763.)

In 1904, milk samples representing 22 counties in England were taken at the railway station and submitted to Doctor Klein, with the result that out of 39 samples tubercle bacilli were found in 3; in August, 1905, a second series representing 22 counties, and out of 22 samples 2 contained tubercle bacilli; in 1906, a third series, representing 13 counties, yielded 2 positive tuberculous milks out of 25 samples.

Proskauer, Seligmann, and Croner. Zeit. Hyg., Bd. 57, 1907, p. 173.

Made an examination of the milk sent in from Denmark, comparing it with Berlin milk. The examination was very thorough, including a search for tubercle bacilli by means of animal inoculation. Danish milk: Thirteen specimens examined, 5 found to contain tubercle bacilli (38.5 per cent). There appears to have been a verbal agreement with the contracting parties that the milk furnished should have been heated 80° to 84° C. Berlin milk: Of 9 samples, 5 contained tubercle bacilli (55.5 per cent). However, in five tests of milk from dairies controlled by veterinary inspections no specimens were found to contain tubercle bacilli.

Hess, Alfred H. The incidence of tubercle bacilli in New York City milk, with a study of its effects on a series of children. J. A. M. Ass., Vol. LII, No. 13. (19 .)

One hundred and twelve specimens of raw milk were examined by inoculation into 224 guinea pigs of the cream and sediment obtained by centrifugalization, but in 5 instances the animals died within two weeks, or were lost in other ways, leaving only 107 samples to be considered.

There were 17 positive results out of the 107, which means that 16 per cent contained tubercle bacilli.

THE NUMBER OF TUBERCULAR COWS IN THE DAIRIES SUPPLYING WASHINGTON, D. C.

A letter was addressed to Dr. W. C. Woodward, health officer, Washington, D. C., and to the Agricultural Department, requesting data as to the number of cows in dairies supplying milk to the city of

Washington that had responded to the tuberculin test. Dr. J. R. Mohler stated October 4, 1907, that of 1,147 recently tested cows supplying milk to the city of Washington, 214, or 18.6 per cent, responded to the tuberculin test. He stated that he did not consider this a fair estimate of the extent of tuberculosis in the dairy herds of this vicinity as the tests were only being applied to those herds which had recently been cleansed by private tests or appear so healthy that their owners have no fear of having them tested.

I am informed by the District health department that 1,059 cows, from 51 herds in Virginia, Maryland, and the District of Columbia, supplying milk to the city of Washington were tested for their reaction to tuberculin; of this number 160, or 15.1 per cent of the total number of cows tested, responded to the tuberculin test.

Of course the above figures furnished by the Department of Agriculture and the District health department do not give a fair idea of the prevalence of tuberculosis in the herds supplying milk to Washington, as only the owners of those herds who had reason to think that their cows were free from tuberculosis permitted the test to be made. If the test had been applied to all the cows supplying milk to the District, I have no doubt that the percentage would be very much higher than the above figures would seem to indicate.

RESULTS OF TUBERCULIN TESTS ELSEWHERE THAN IN HERDS SUPPLYING WASHINGTON.

The following figures by Salmon[a] show the number and percentage of cattle carcasses condemned for tuberculosis during the years 1901–1905 in the meat-inspection service of the Bureau of Animal Industry:

Year.	Number examined.	Per cent condemned.
1901	5,219,149	0.10
1902	5,559,969	.14
1903	6,134,410	.14
1904	6,350,011	.16
1905	6,096,597	.18

This does not show the total number of animals affected with tuberculosis, for in many cases only a part of the carcass was condemned and probably many had the disease so slightly that the entire carcass was passed as fit for food.

The following table, also taken from Salmon's article, showing the results of the tuberculin test of cattle in some States, is of value as showing the wide distribution of bovine tuberculosis. It must be remembered that most of the herds tested were suspected herds, which may account for the very high percentages found.

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[a] Salmon, D. E.: Bull. No. 38, Bureau Animal Industry, 1906.

Results of the tuberculin tests of cattle in various States.

State.	Number tested.	Number tuberculous.	Per cent tuberculous.
Vermont	60,000	2,390	3.9
Massachusetts	24,685	12,443	50.0
Massachusetts, entire herds	4,093	1,080	26.4
Connecticut	6,300	....	14.2
New York, 1894	947	66	6.9
New York, 1897-98	1,200	163	18.4
Pennsylvania	34,000	4,800	14.1
New Jersey	2,500	....	21.4
Illinois, 1897-98	929	....	12.0
Illinois, 1899	3,655	560	15.3
Michigan	....	....	13.0
Minnesota	3,430	....	11.1
Iowa	873	122	13.8
Wisconsin			
Experiment station tests—			
Suspected herds	323	115	35.6
Nonsuspected herds	935	84	9.
State veterinarian's tests—			
Suspected herds	588	191	32.5
Tests of local veterinarians under State veterinarian on cattle intended for shipment to States requiring tuberculin certificate	3,421	76	2.2

THE CHARACTERISTICS OF RABINOWITSCH'S BUTTER BACILLUS.

The results of some of the earlier workers are open to criticism in view of Rabinowitsch's discovery of an acid-fast bacillus in butter morphologically similar to the tubercle bacillus. If guinea pigs are inoculated with milk or butter containing the acid-fast butter bacillus they may often die and will present lesions to the naked eye very similar to those produced by the tubercle bacillus. For that reason I give the following description of the cultural characteristics and post-mortem appearances caused by this organism taken from Annett's[a] article.

The characteristics of Rabinowitsch's micro-organism are as follows: It is immotile, and in form closely resembles the bacillus tuberculosis. The bacilli generally occur singly and are often slightly curved; but when growing rapidly in tissue bacilli are often found lying parallel. Sometimes they form long unbranched threads and sometimes are divided into short pieces. The bacilli are somewhat thicker than the tubercle bacillus and often show a club-shaped swelling on one side. Spores are not formed, but one portion of the bacillus stains often more intensely than the rest. The bacilli stained by many methods of staining tubercle bacilli can not be distinguished from the latter: only by the employment of very dilute watery solutions of methylene blue could any distinguishing feature be observed, viz, that bacilli from a culture of bacillus tuberculosis stain only at one spot, the rest of the bacillus remaining unstained; while in the case of bacillus pseudo-tuberculosis the whole bacillus stains faintly and generally uniformly, seldom showing a more deeply stained part.

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[a] Annett, H. E.: Tubercle bacilli in milk, butter, and margarine. Report Thompson Yates Laboratory, 1898–99, pp. 29–35.

Cultural differences, however, occur. On agar, the bacilli taken direct from an infected animal produce visible colonies on the second or third day. At first the agar surface is covered with a thick, moist, creamy layer; in old cultures by a folded membrane often orange or copper colored. After repeated passages through animals cultures on agar or glycerin-agar show a dry, brittle, crumpled membrane resembling that of bacillus tuberculosis. In plate cultures the deep colonies are gray in color, round or oval, and uniformly granular. On the surface, colonies are better developed, have a uniform granular gray center, and a clear, wavy outer zone. The surface of the colony is often dry and conical. On butter-agar in fresh cultures the colonies are small, white, and dry, later spreading over the whole surface and becoming yellow or copper colored. On potato a luxuriantly growing, moist, gray layer is formed. In gelatin, growth proceeds very slowly at ordinary room temperatures, colonies becoming visible on the third day. In broth, and especially in glycerin broth, growth is rapid, forming in two or three days a folded membrane on the surface, the broth remaining clear, the culture closely resembling that of bacillus tuberculosis. Broth cultures are distinguishable from those of bacillus tuberculosis by their characteristic odor, being unpleasant and ammoniacal; that of bacillus tuberculosis being agreeable and resembling the odor of flowers. A small quantity of indol is formed in broth cultures, which is not so in bacillus tuberculosis cultures. Milk is not coagulated, and on the surface is an abundant yellowish-red layer which clings firmly to the glass. On albumin-free colorless media a growth appears in two or three days, becoming in ten days a thick, yellow, folded membrane; bacillus tuberculosis in the same time on such media forming a thin layer just covering the surface and just beginning to fold. The presence of fat in these bacilli can easily be demonstrated, as in the case of bacillus tuberculosis.

PATHOGENIC PROPERTIES OF BACILLUS PSEUDO-TUBERCULOSIS.

The following are the post-mortem appearances observed in a guinea pig killed three or four weeks after intraperitoneal injection of butter containing the bacillus pseudo-tuberculosis: There is a slightly distended abdomen; also peritonitis, with adhesions varying in nature from delicate fibrinous bands to firm connective tissue.

The peritoneum and mesentery are studded with nodules. The mesenteric glands are swollen and may contain purulent or caseous matter. The liver is covered with nodules and patches which may be raised above the liver substance or may penetrate into the liver parenchyma. The spleen is sometimes only enlarged; at other times thickly studded with nodules. The kidneys show yellowish patches. The lungs are covered with small transparent nodules which do not penetrate into the lung tissue. The sternal lymphatic glands are swollen, but show no caseation. Numerous bacilli can be demonstrated in these lesions. Many animals after injection show considerable signs of illness during the first fourteen days, with diminution in weight, and then recover. Pure cultures of this bacillus are only pathogenic for guinea pigs (but not always); rabbits and white mice are immune. After intraperitoneal inoculation of guinea pigs some die in from four to eight weeks, with considerable emaciation, and show the following post-mortem appearances: At the seat of inoculation there is a purulent infiltration containing the characteristic bacilli; also peritonitis, varying in intensity from a flocculent fibrinous exudation to strong connective tissue adhesions. The mesentery is studded with small nodules; the glands are enlarged, but not caseous; there are patches on the liver, and miliary nodules throughout an enlarged spleen. The thoracic cavity and organs are often almost exempt from lesions. Infected animals do not react to tuberculin. Histologically the nodules in the liver and spleen consist of a collection of lymphoid elements with but very few epithelioid and multinuclear cells. The bacilli are found in the middle of a young nodule, toward the periphery if caseation has commenced. The typical giant cells of tuberculosis do not occur. More often--especially after inoculation with butter containing the pseudo-tuberculosis bacilli—the nodules appear to consist of a central necrosed portion surrounded by a leucocytic infiltrated area.

COLLECTION OF SAMPLES AND TECHNIC.

The samples of milk were all collected and brought to the Hygienic Laboratory by an inspector of the health department of the District of Columbia. Usually a pint bottle, though sometimes a quart, with the paper cap untampered with was obtained either from the diary or delivery wagon. The bottle was at once placed on ice by the collector and usually reached the laboratory in about one hour after collection. A few samples were obtained from some of the hospitals and charitable institutions of the District. The milk and cream were well mixed by vigorously shaking the bottle. The sample for plating was taken out with a sterile pipette, and then 50 cubic centimeters of the mixed milk was put into a large sterile centrifuge flask. To the 50 cubic centimeters of milk was added 100 cubic centimeters of sterile water. The flask was then put into the centrifuge machine and centrifuged for one hour at about 2,000 revolutions per minute. The milk was diluted with twice its volume of water with the idea that it would decrease the specific gravity of the milk and so permit of the easier sedimentation of the tubercle bacilli. Usually only one animal was inoculated from each sample, though in some cases two animals were used. Guinea pigs, largely those raised in the laboratory, of as uniform weight as obtainable, were inoculated with 5 cubic centimeters of the sediment of this centrifugalized mixture of milk and water. The inoculation was made subcutaneously in the belly wall. For each guinea pig a different syringe was used. All of the guinea pigs, usually 8, that being the usual number of daily samples, inoculated on the same day were kept in the same cage, those that remained healthy being controls on their environment, etc. The guinea pigs were examined for enlarged glands after about four weeks, and those with enlarged glands were separated from the others so as to avoid the danger of infecting others if the glands broke down.

Many of the animals inoculated died from acute infection with the millions of other bacteria in the milk. Autopsies were made on all the animals that died, but no attempt was made to determine the causal organisms other than the tubercle bacillus.

Those guinea pigs which did not die in at least two months were chloroformed, after having been tested with tuberculin, and careful autopsies were made on each animal. Smears, cultures, and sections were made from the various organs of the animals that showed any change from the normal. The smears were stained with carbol-fuchsin and examined for acid-fast bacilli. Cultures were made on glycerinized potato and glycerin-agar. In no instance did any of the cultures show a quick-growing acid-fast organism resembling in any way Rabinowitch's butter bacillus. The sections were stained with carbol-fuchsin for tubercle bacilli, and also with haemalum and eosine for histological appearances. The above details were carried out with few exceptions in all of the animals that gave a positive result.

It occurred to me that those animals which had tuberculosis might be differentiated from those with other infections by giving all of the guinea pigs alive at the end of two months a sufficient dose of tuberculin to cause the death of the tuberculous animals in less than twenty-four hours. Several preliminary tests on known tubercular animals showed that 2 cubic centimeters of crude tuberculin given subcutaneously would almost invariably cause the death of such a guinea pig in from six to eighteen hours. As high as 7 cubic centimeters of the same tuberculin given to a healthy pig caused only a temporary discomfort, passing off in a few hours. A rather hasty search of the literature failed to show that this idea of giving an amount of tuberculin sufficient to cause the death of a tubercular animal as a means of differentiating true tuberculosis from infection with other acid-fast organisms had ever been used by previous workers. The febrile reaction in a sick guinea pig on account of the great variation in the temperature of the animal from handling, etc., is too variable a factor, and a more definite reaction, such as the death of the animal, is necessary. The technic was as follows: All of the animals, in lots of about 30, were given early in the morning 2 cubic centimeters of the tuberculin subcutaneously; they were closely watched and as soon as an animal appeared sick it was placed aside; as soon after death as possible the animal was autopsied; smears, cultures, and sections were made. Of all the guinea pigs, about 250, that received the tuberculin, no animal that did not have tuberculosis died. Two or three that had slight lesions did not die, but became sick. It was noted that all of the animals died whose lesions had caseated. The reaction, I think, was of distinct service in eliminating infections with other acid-fast organisms. The suggestion is made that with some modification the procedure may have a distinct place as an aid in differentiating true tuberculosis from infections with other acid-fast organisms which produce tubercular-like lesions.

Samples of milk were examined from 104 different dairies; 10 samples from 7 hospitals and asylums are also included in this number, they being charged also to the dairy supplying the milk.

The following tables show the laboratory number of the dairy, where collected, date of collection, whether the guinea pig inoculated died or was killed, interval between inoculation and death, and results of the autopsy.

It is interesting to note that where 2 guinea pigs were inoculated with the same sample of milk, in two instances both animals showed tuberculosis and in two instances only one was positive:

TABLE No. 1.

No. of guinea pig.	Weight.	Date of inoculation.	Source.	Dairy.	Result	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
44		July 22	Wagon.	No. 1	Died	20	No evidence of tubercle.		
132		Aug. 5	Dairy	do	do	2			
230		Aug. 19	do	do	Killed	72	Negative		
231		do	do	do	Died	4			
14		July 15		No. 2	Killed	101	Negative		
100		July 30	Providence Hospital.	do	do	86	do		
101		do	do	do	Died	27	No evidence of tubercle.		
329		Aug. 30	do	do	Killed	63	Negative		

TABLE No. 1—Continued.

No. of guinea pig.	Weight.	Date of inoculation.	Source.	Dairy.	Result.	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
330	......	Aug. 30	Providence Hospital.	No. 2	Killed	63	Negative		
281	......	Aug. 26	Dairy	No. 3	do	61	do		
282	......	do	do	do	do	61	do		
114	......	Aug. 1	do	No. 4	do	88	do		
299	......	Aug. 27	do	do	Died	5			
300	......	do	do	do	Killed	95	Negative		
2	......	July 12		No. 5	Died	2			
155	......	Aug. 7	Dairy	do	do	8			
156	......	do	do	do	Killed	82	Negative		
61	......	July 24	do	No. 6	do	93	do		
86	......	July 29	Wagon	do	do	90	do		
256	......	Aug. 22	do	do	do	70	do		
143	......	Aug. 6	do	No. 7	do	84	do		
17	......	July 16	do	No. 8	do	100	do		
122	......	Aug. 2	do	do	do	87	do		
7	......	July 12		No. 9	do	104	do		
48	......	July 22		do	Died	21	No evidence of tubercle.		
195	......	Aug. 14	Dairy	do	do	57	do		
196	......	do	do	do	Killed	77	Negative		
303	......	Aug. 28	do	do	do	65	do		
163	......	Aug. 8	do	No. 10	Died	81	do		
239	......	Aug. 20		do	do	57	No evidence of tubercle.		
51	......	July 23	Wagon	No. 11	Killed	94	Negative		
64	......	July 24	Dairy	do	do	94	do		
68	......	July 25	Wagon	do	do	92	do		
78	......	July 26	Children's Hospital.	do	Died	3			
85	......	do	do	do	Killed	92	Negative		
119	......	Aug. 1	Columbia Hospital.	do	Died	1½			
123	......	Aug. 2	do	do	Killed	77	Negative		
131	......	Aug. 5	Wagon	do	Died	1½			
226	......	Aug. 19	do	do	Killed	72	Negative		
27	......	July 17		No. 12	Died	19	No evidence of tubercle.		
151	......	Aug. 7	Dairy	No. 13	Killed	83	Negative		
97	......	July 30		No. 14	do	88	do		
105	......	July 31	Wagon	No. 15	do	89	do		
146	......	Aug. 7	do	do	Died	4			
93	......	July 29	Dairy	No. 16	Killed	89	Inguinal and retroperitoneal glands caseous; mediastinal glands enlarged; spleen enlarged and studded with tubercles; liver and lung numerous tubercular foci; sections show histological tubercles and tubercle bacilli.		
41	......	July 19		No. 17	do	98	Negative		

TABLE No. 1 Continued.

No. of guinea pig.	Weight.	Date of inoculation.	Source.	Dairy.	Result.	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
57		July 23		No. 18	Killed	93	Negative		
144		Aug. 6	Dairy	do	do	83	do		
237		Aug. 20	Wagon	do	do	69	Inguinal and retroperitoneal glands enlarged and caseous; spleen and liver enlarged and studded with tubercles; lungs contain tubercular foci; sections show histological tubercles and tubercle bacilli.	+	
22		July 16		No. 19	Died	66	No evidence of tubercle.		
76		July 25	Dairy	do	Killed	92	Negative		
191		Aug. 13	do	do	Died	3			
323		Aug. 30	do	do	Killed	63	Negative		
25		July 17		No. 20	do	100	do		
70		July 25		do	do	93	do		
88		July 29	Dairy	do	Died	33	No evidence of tubercle.		
179		Aug. 12		do	Killed	68	Negative		
274		Aug. 23	Dairy	do	do	69	do		
20		July 16		No. 21	do	100	do		
125		Aug. 2	Dairy	do	do	97	do		
247		Aug. 21	do	do	Died	1½	do		
297		Aug. 27	Wagon	do	Killed	65	do		
298		do	do	do	do	65	do		
321		Aug. 30	do	do	Died	40	No evidence of tubercle.		
39		July 19	do	No. 22	Killed	98	Negative		
102		July 30	do	do	do	88	do		
188		Aug. 13	do	do	Died	20	No evidence of tubercle.		
108		July 31	do	No. 23	Killed	89	Negative		
21		July 16		No. 24	do	100	do		
124		Aug. 2	Wagon	do	do	87	do		
166		Aug. 9	Dairy	do	do	81	do		
248		Aug. 21	do	do	Died	2			
325		Aug. 30	Wagon	do	do	41	No evidence of tubercle.		
326		do	do	do	do	2			
107		July 31		No. 25	Killed	89	Negative		
262		Aug. 22	Dairy	do	do	70	do		
279		Aug. 26	do	do	do	66	do		
169		Aug. 9	Wagon	No. 26	do	80	do		
10		July 15	do	No. 27	Died	5			
52		July 23	do	do	Killed	94	Negative		
53		do	do	do	do	93	do		
145		Aug. 7	do	do	do	83	do		
263		Aug. 22	do	do	do	83	do		
28		July 17		No. 28	Died	4	do		
73		July 25		do	Killed	93	do		
74		do		do	Died	35	No evidence of tubercle.		
143		Aug. 7	Dairy	do	Killed	83	Negative		
319		Aug. 29	do	do	do	63	do		

TABLE No. 1—Continued.

No. of guinea pig.	Weight.	Date of inoculation.	Source.	Dairy.	Result.	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
320		Aug. 29	Dairy	No. 28	Killed	63	Negative		
40		July 19		No. 29	do	98	do		
106		July 31		do	do	89	do		
261		Aug. 22	Dairy	do	do	70	do		
31		July 18	Wagon	No. 30	do	99	do		
258		Aug. 22	do	do	do	70	do		
98		July 30		No. 31	do	82	do		
12		July 15		No. 32	do	101	do		
67		July 24		do	Died	1½			
178		Aug. 12	Dairy	do	Killed	83	Negative		
29		July 17		No. 33	Died	3			
71		July 25		do	Killed	93	Inguinal, axillary and retroperitoneal glands caseous; spleen enlarged, studded with tubercles; liver and lung contained many tubercles; sections show histological tubercles and tubercle bacilli.	+	
72		do		do	do	93	Negative		
171		Aug. 9	Dairy	do	Died	24	No evidence of tubercle.		
232		Aug. 19	do	do	do	4			
233		do	do	do	do	2			
111		Aug. 1	Wagon	No. 34	Killed	88	Negative		
211		Aug. 15	Dairy	do	do	76	do		
311		Aug. 29	Wagon	do	do	63	do		
121		Aug 2	do	No. 35	do	87	do		
238		Aug. 20	do	do	do	70	do		
260		Aug. 22	do	No. 36	do	70	do		
216		Aug. 16	Dairy	No. 37	do	76	do		
113		Aug. 1	Wagon.	No. 38	do	88	do		
142		Aug 6	do	do	do	84	do		
229		Aug. 19	do	do	Died	14			
296		Aug. 27	do	do	Killed	65	Negative		
15		July 15	do	No. 39	do	101	do		
94		July 29	Dairy	do	Died	5			
223		Aug. 19	Wagon	do	Killed	73	Negative		
224		do	do	do	Died	11			
283		Aug 26	Dairy	do	Killed	61	Negative		
284		do	do	do	do	61	do		
138		Aug 6	do	No. 40	do	84	do		
315		Aug. 29	do		do	63	do		
32		July 18		No. 41	do	99	do		
66		July 24		do	do	93	do		
79		July 26	Wagon	do	do	92	do		
174		Aug 12	do	do	Died	1½			
234		Aug. 20	do	do	Killed	71	Negative		
235		do	do	do	do	71	do		
207		Aug. 15	Dairy	No. 42	do	76	do		
24		July 17		No. 43	do	100	do		

TABLE No. 1—Continued.

No. of guinea pig.	Weight.	Date of inoculation.	Source.	Dairy.	Result.	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
128		Aug. 5	Dairy	No. 4,	Killed	84	Negative		
295		Aug. 27	Wagon	do	do	65	do		
23		July 17	do	No. 44	do	100	do		
177		Aug. 12	do	do	Died	70	do		
244		Aug. 21	do	do	Killed	71	do		
245		do	Georgetown Hospital.	do	do	69	do		
246		do	do	do	do	69	do		
322		Aug. 30	Wagon	do	do	62	do		
6		July 12		No. 45	do	104	do		
83		July 26		do	Died	11	do		
172		Aug. 12	Dairy	do	do	1½	do		
173		do	do	do	Killed	42	Inguinal glands caseous; axillary and mediastinal glands enlarged; spleen enlarged and studded with tubercles; few foci in liver; sections show histological tubercles and tubercle bacilli.	-	
265		Aug. 23	do	do	Died	4			
266		do	do	do	do	62	Inguinal, axillary and retroperitoneal glands enlarged and caseous; spleen and liver enlarged and studded with tubercles; lungs contain many tubercle foci; sections show histological tubercles and tubercle bacilli.	+	
267		do	do	do	Killed	68	Inguinal, retroperitoneal and mediastinal glands enlarged and caseous; liver and spleen enlarged and studded with tubercles; sections show histological tubercles and tubercle bacilli.	+	
159		Aug. 9	Wagon	No. 46	do	82	Inguinal glands enlarged; spleen greatly enlarged, contains numerous tubercles; few tubercles in liver; sections show histological tubercles.	+	
268		Aug. 23	do		do	69	Negative		
75		July 25		No. 47	Died	17	No evidence of tubercle.		
291		Aug. 27	Wagon	do	Killed	65	Negative		

TABLE No. 1 Continued.

No. of guinea pig.	Weight.	Date of inoculation.	Source.	Dairy.	Result.	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
292		Aug. 27	Wagon	No. 47	Killed	65	Negative		
120		Aug. 2	do	No. 48	do	87	do		
4		July 12		No. 49	do	104	do		
222		Aug. 16	Dairy	do	do	75	do		
307		Aug. 28	Wagon	do	do	64	do		
18		July 16	do	No. 50	do	96	do		
236		Aug. 20	do	do	do	71	do		
168		Aug 9	do	No. 51	do	81	do		
324		Aug. 30	do	do	do	62	do		
77		July 25	do	No. 52	Died	3			
180		Aug. 12	do	do	do	3			
181		Aug. 12	do	do	Killed	71	Negative		
43		July 22		No. 53	Died	22	No evidence of tubercle		
65		July 23		do	Killed	93	Negative		
89		July 29		do	do	90	do		
152		Aug. 7	Dairy	do	do	83	do		
182		Aug. 12	Garfield Hospital.	No. 54	do	83	do		
183		do	do	do	do	83	do		
192		Aug. 13	do	do	do	78	do		
46		July 22		No. 55	Died	4			
149		Aug. 7	Dairy	do	do	1½			
150		do	do	do	Killed	83	Negative		
270		Aug. 23	do	do	do	69	do		
271		do	do	do	do	69	Anterior mediastinal glands enlarged and caseous; liver studded with numerous tubercles; tubercle bacilli found in smears.	+	
80		July 26	Wagon	No. 56	do	92	Negative		
273		Aug. 23	Dairy	do	do	69	do		
165		Aug. 9	Wagon	No. 57	do	81	do		
54		July 23	do	No. 58	do	94	do		
251		Aug. 21	do	do	do	71	do		
63		July 24		No. 59	do	93	do		
208		Aug. 15	Dairy	No. 60	do	76	do		
90		July 29	Wagon	No. 61	do	89	do		
293		Aug. 27	do	do	do	65	do		
294		do	do	do	do	65	do		
197		Aug. 14	do	No. 62	do	77	Spleen enlarged and studded with tubercles; liver contains tubercular foci; spleen sections show histological tubercles and tubercle bacilli.	+	
164		Aug 9	do	No. 63	do	81	Negative		
167		do	do	No. 64	do	81	do		
243		Aug. 20	do	do	do	71	do		
99		July 30	do	No. 65	Died	1½			
313		Aug. 29	do	do	Killed	63	Negative		

TABLE No. 1—Continued.

No. of guinea pig	Weight	Date of inoculation.	Source.	Dairy.	Result.	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
314		Aug. 29	Wagon	No. 65	Died	15			
139		Aug. 6	Dairy	No. 66	Killed	84	Negative		
306		Aug. 28	do	do	do	64	do		
272		Aug. 23	Wagon	No. 67	do	66	do		
36		July 18		No. 68	Died	3			
91		July 29	Dairy	do	Killed	89	Negative		
92		do	do	do	do	89	do		
285		Aug. 26	do	do	do	66	do		
286		do	do	do	do	66	do		
170		Aug. 9	do	No. 69	do	81	do		
309		Aug. 28	do	do	do	64	do		
317		Aug. 29	do	do	Died	1			
328		Aug. 30	do	do	Killed	63	Negative		
11		July 15	Wagon	No. 70	do	101	do		
160		Aug. 8	do	do	do	82	do		
257		Aug. 22	do	do	Died	1			
199		Aug. 14	Dairy	No. 71	Killed	76	Negative		
137		Aug. 6	Wagon	No. 72	do	84	do		
215		Aug. 16	do	do	do	75	do		
16		July 16	do	No. 73	Died	1½	do		
55		July 23	do	do	do	3	do		
56		do	do	do	Killed	95	Inguinal and retroperitoneal glands caseous; spleen enlarged, studded with tubercles; liver and lung contained many tubercles; sections show histological tubercles and tubercle bacilli.	+	
161		Aug. 8	Dairy	do	do	82	Negative		
162		do	do	do	do	82	do		
240		Aug. 20	do	do	do	71	do		
289		Aug. 27	Wagon	do	Died	1½	do		
290		do	do	do	do	64	Inguinal and mediastinal glands enlarged and caseous; liver studded with tubercles; sections liver and glands show histological tubercle and tubercle bacilli; other organs lost.	+	
1		July 12		No. 74	Killed	104	Negative		
158		Aug. 8	Dairy	do	Died	1½			
225		Aug. 19	do	do	do	26	No evidence of tubercles		
133		Aug. 5	Wagon	No. 75	Killed	84	Negative		
228		Aug. 19	do	do	do	72	do		
87		July 29	do	No. 76	do	89	do		
221		Aug. 16		do	do	75	do		
13		July 15		No. 77	do	101	do		
218		Aug. 16	Wagon	do	Died	1½			

TABLE No. 1—Continued.

No. of guinea pig.	Weight.	Date of inoculation.	Source.	Dairy.	Result.	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
202		Aug. 14	Wagon	No. 78	Killed	77	Negative		
33		July 18		No. 79	Died	84	No evidence of tubercle		
69		July 25	Wagon	No. 80	Killed	93	Negative		
176		Aug. 12	do	do	do	78	do		
5		July 12		No. 81	do	104	do		
82		July 26		do	do	92	do		
175		Aug. 12	Dairy	do	do	78	do		
212		Aug. 15	Sibley Hospital	do	do	76	do		
213		do	do	do	do	76	do		
264		Aug. 23	Dairy	do	Died	19			
312		Aug. 29	Wagon	do	Killed	63	Negative		
45		July 22		No. 82	Died	19			
129		Aug. 5	Wagon	do	Killed	84	Negative		
184		Aug. 13	Dairy	do	do	79	do		
185		do	do	do	Died	20			
241		Aug. 20	do	do	Killed	71	Negative		
242		do	do	do	do	71	do		
310		Aug. 28	do	do	do	64	do		
318		Aug. 29	do	do	Died	1½			
327		Aug. 30	do	do	Killed	63	Negative		
96		July 30	Wagon	No. 83	do	89	do		
34		July 18		No. 84	Died	4			
103		July 31	Orphan asylum	do	do	81	Inguinal, retroperitoneal, and mediastinal glands caseous; spleen enlarged, studded with tubercles; liver and lungs contain numerous tubercle foci; sections show histological tubercles and tubercle bacilli.	+	
104		do	do	do	Killed	89	Inguinal and retroperitoneal glands enlarged and caseous; spleen and liver enlarged and studded with tubercles; sections show histological tubercles and tubercle bacilli.	+	
130		Aug. 5	Wagon	do	do	83	do	+	
147		Aug. 7	do	do	do	83	Negative		
186		Aug. 13	Dairy	do	Died	2			
187		do	do	do	Killed	79	Negative		
275		Aug. 26	do	do	do	66	Inguinal and mediastinal glands enlarged and caseous; spleen and liver enlarged and studded with tubercles; many tubercle bacilli in smears.	+	

TABLE No. 1—Continued.

No. of guinea pig.	Weight.	Date of inoculation.	Source.	Dairy.	Result.	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
276		Aug. 26	Wagon	No. 84	Died	16			
198		Aug. 14	do	No. 85	Killed	77	Negative		
304		Aug. 28	do	do	do	64	do		
140		Aug. 6	do	No. 86	do	84	Inguinal glands enlarged and caseous; retroperitoneal and mediastinal glands enlarged; spleen and liver enlarged and studded with numerous tubercles; tubercle in liver; sections show histological tubercles.	+	
206		Aug. 15	do	do	do	76	Negative		
305		Aug. 28	do	do	do	64	do		
109		July 31	do	No. 87	do	79	do		
280		Aug. 26	do	do	do	66	do		
227		Aug. 19	Wagon	No. 88	do	73	do		
301		Aug. 27	do	do	do	65	do		
302		do	do	do	do	65	do		
259		Aug. 22	do	No. 89	do	70	do		
38		July 19	do	No. 90	Died	3			
95		July 30	do	No. 91	Killed	88	Negative		
209		Aug. 15	Dairy	do	do	76	do		
30		July 17		No. 92	Died	5			
135		Aug. 5	Dairy	do	Killed	84	Negative		
136		do	do	do	Died	2			
254		Aug. 21	do	do	Killed	69	Negative		
255		do	do	do	Died	2			
84		July 26		No. 93	Killed	92	Negative		
141		Aug. 6	Dairy	do	do	84	do		
269		Aug. 23	do	do	do	69	do		
203		Aug. 14	Wagon.	No. 94	do	77	do		
8		July 12		No. 95	do	104	do		
42		July 19		do	do	98	do		
49		July 22		do	Died	4			
58		July 23		do	Killed	94	Negative		
134		Aug. 5	Wagon	do	do	84	do		
151		Aug. 8	do	do	do	82	do		
201		Aug. 14	Dairy	do	Died	3			
214		Aug. 16	do	do	do	1½			
3		July 12		No. 96.	Killed	104	Negative		
157		Aug. 8	Dairy	do	do	82	do		
210		Aug. 15	do	do	do	76	do		
19		July 16	Wagon.	No. 97	do	101	do		
127		Aug. 2	do	do	do	80	do		
35		July 18		No. 98.	do	99	do		
60		July 24	Wagon.	do	Died	3			
189		Aug. 13	Dairy	do	Killed	76	Negative		
277		Aug. 26	do	do	Died	8			

TABLE No. 1—Continued.

No. of guinea pig.	Weight.	Date of inoculation.	Source.	Dairy.	Result.	Days since inoculated.	Autopsy.	Tubercle bacilli.	
								Present.	Absent.
278		Aug. 26	Dairy	No. 98	Killed	66	Negative		
126		Aug. 2	do	No. 99	do	87	do		
190		Aug. 13	do	do	Died	4			
249		Aug. 21	do	do	do	2			
250		do	do	do	do	5			
47		July 22		No. 100	do	18			
148		Aug. 7	Dairy	do	Killed	83	Negative		
219		Aug. 16	do	do	do	75	do		
220		do	do	do	do	75	do		
112		Aug. 1	Wagon	No. 101	do	87	do		
26		July 17		No. 102	do	100	do		
62		July 24	Wagon	do	do	95	do		
117		Aug. 1	Dairy	do	Died	9			
193		Aug. 13	do	do	do	25	No evidence of tubercle		
194		do	do	do	Killed	75	Negative		
59		July 23		No. 103	do	94	Inguinal and retroperitoneal glands caseous; spleen enlarged and studded with tubercles; liver showed numerous tubercular foci; sections show histological tubercles and tubercle bacilli.	+	
200		Aug. 14	Dairy	do	do	77	Negative		
308		Aug. 28	do	do	do	64	do		
316		Aug. 29	do	do	Died	2			
9		July 15		No. 104	do	12			
37		July 19		do	do	3			
252		Aug. 21	Wagon	do	Killed	71	Negative		
253		do	do	do	do	71	do		
287		Aug. 26	do	do	do	67	do		
288		do	do	do	Died	19			

The following table gives a summary of the above protocols. It shows the laboratory number of the dairy, number of samples from each dairy, number of samples lost by the animal dying in less than three weeks of other infections, number samples remaining for observation, and total number of samples for each dairy positive for tuberculosis.

The same details are shown in Table 3 for the milk collected from the charitable institutions.

TABLE NO. 2.

Dairy.	Number of samples.	Number samples lost by acute death of guinea pig	Number samples remaining.	Number samples positive for tuberculosis.
1	3	2	1	0
2	3	0	3	0
3	1	0	1	0
4	2	0	2	0
5	2	1	1	0
6	3	0	3	0
7	1	0	1	0
8	2	0	2	0
9	4	1	3	0
10	2	0	2	0
11	8	2	6	0
12	1	1	0	0
13	1	0	1	0
14	1	0	1	0
15	2	1	1	0
16	1	0	1	1
17	1	0	1	0
18	3	0	3	1
19	4	1	3	0
20	5	0	5	0
21	5	1	4	0
22	3	1	2	0
23	1	0	1	0
24	5	1	4	0
25	3	0	3	0
26	1	0	1	0
27	4	1	3	0
28	4	1	3	0
29	3	0	3	0
30	2	0	2	0
31	1	0	1	0
32	3	1	2	0
33	4	2	2	1
34	3	0	3	0
35	2	0	2	0
36	1	0	1	0
37	1	0	1	0
38	4	1	3	0
39	4	1	3	0
40	2	0	2	0
41	5	1	4	0
42	1	0	1	0
43	3	0	3	0
44	5	0	5	0
45	4	1	3	2
46	2	0	2	1
47	2	1	1	0
48	1	0	1	0
49	3	0	3	0
50	2	0	2	0
51	2	0	2	0
52	2	1	1	0
53	4	1	3	0
54	2	0	2	0

TABLE NO. 2—Continued.

Dairy.	Number of samples.	Number samples lost by acute death of guinea pig.	Number samples remaining.	Number samples positive for tuberculosis.
55	3	1	2	1
56	2	0	2	0
57	1	0	1	0
58	2	0	2	0
59	1	0	1	0
60	1	0	1	0
61	2	0	2	0
62	1	0	1	1
63	1	0	1	0
64	2	0	2	0
65	2	1	1	0
66	2	0	2	0
67	1	0	1	0
68	3	1	2	0
69	4	1	3	0
70	3	1	2	0
71	1	0	1	0
72	2	0	2	0
73	5	1	4	2
74	3	1	2	0
75	2	0	2	0
76	2	0	2	0
77	2	1	1	0
78	1	0	1	0
79	1	0	1	0
80	2	0	2	0
81	6	1	5	0
82	7	2	5	0
83	1	0	1	0
84	6	1	5	3
85	2	0	2	0
86	3	0	3	1
87	2	0	2	0
88	2	0	2	0
89	1	0	1	0
90	1	1	0	0
91	2	0	2	0
92	3	1	2	0
93	3	0	3	0
94	1	0	1	0
95	8	3	5	0
96	3	0	3	0
97	2	0	2	0
98	4	1	3	0
99	3	2	1	0
100	3	1	2	0
101	1	0	1	0
102	4	1	3	0
103	4	1	3	1
104	4	2	2	0
Total....	272	49	223	15
Per cent		18	82	6.72

TABLE NO. 3.

Hospital	Number of samples.	Number samples lost by acute death of guinea pig.	Number samples remaining.	Number samples positive for tuberculosis.
Providence	2	0	2	0
Children's	1	0	1	0
Georgetown	1	0	1	0
Garfield	2	0	2	0
Sibley	1	0	1	0
Orphan Asylum	1	0	1	[a] 1
Columbia	2	1	1	0
Total	10	1	9	1
Per cent	...	10	90	11.1

[a] Both pigs from sample positive.

RÉSUMÉ.

It will be seen from the above that of 272 samples of milk 49, or 18 per cent of the samples, were lost by the animals dying in less than three weeks and before sufficient time had elapsed for them to develop tuberculosis. Attention is invited to the fact that the milk from some of the dairies killed acutely a high percentage of all of the animals to which it was given.

Of the 272 samples 223, or 82 per cent, remained for study.

Of the 223 that remained 15, or 6.72 per cent, contained sufficient tubercle bacilli to cause typical tuberculosis in the inoculated animals.

Of the samples of milk from 104 dairies, 2 were lost by acute death of the animals, leaving 102; the milk from 11 of these 102 dairies contained tubercle bacilli. This gives a percentage of 10.7 of the dairies examined showing tubercle bacilli in the milk supplied to their customers.

Ten samples of milk were obtained from 7 charitable institutions of the District; of these 10 samples, 1 was lost by the acute death of the animal, leaving 9 samples from 6 institutions for study. The sample from 1 institution caused tuberculosis in both guinea pigs in which it was inoculated.

These results showing that approximately 11 per cent of the dairies whose milk was examined contained tubercle bacilli virulent for guinea pigs do not, however, give a fair idea of the frequency of the presence of tubercle bacilli in the market milk of the city of Washington. Attention has already been called to the fact that when two animals were inoculated with the same sample both did not always develop tuberculosis; this would indicate that the bacilli are so few in the amount inoculated that one of the animals by being a little more resistant was able to overcome the infection. The amount inoculated, less than 2 cubic centimeters of milk, is a very small portion of a pint bottle. The creamy layer was not inoculated and other workers have shown that tubercle bacilli are more frequent in this than in the bottom milk; it is very probable that if more animals had been inoculated with the same sample and both cream and sediment used the percentage of positive results would have been very much higher. The results, however, as they were found are sufficiently high to emphasize the great necessity for the enactment and rigorous enforcement of a law requiring that all cows supplying milk to the District be tuberculin tested and free of tuberculosis. This test, which is now universally recognized as a means of determining whether an animal has tuberculosis, should be made by a competent veterinarian and those animals that respond should be disposed of in some way so that their milk may no longer be a source of danger to the community.

5. THE RELATION OF GOAT'S MILK TO THE SPREAD OF MALTA FEVER.

THE RELATION OF GOAT'S MILK TO THE SPREAD OF MALTA FEVER.

By John F. Anderson.

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Recently it has been shown that Malta fever is conveyed by means of the milk of goats infected with the specific organism of the disease. While the disease may undoubtedly be spread by other means, the use of infected goat's milk in Malta is by far the most important factor.

Malta fever is a specific febrile infection caused by the *Micrococcus melitensis* discovered by Bruce in 1887. The fever is of an irregular, recurring or undulating type; in a typical case it lasts for several weeks, followed by a period of a few days or weeks of a relative apyrexia, which is again followed by other febrile periods.

Clinically, Malta fever is usually characterized by profuse perspiration, constipation, frequent relapses, often accompanied by pains of a rheumatic or neuralgic character, sometimes swelling of joints or orchitis. The disease is characterized by low mortality and indefinite duration.

Malta fever smolders endemically on the island of Malta, at Gibraltar, and other places on the Mediterranean basin. At times the number of cases at one place constitutes an epidemic. Bruce believes that one attack confers a definite immunity against subsequent attacks. Strangers particularly, visiting in the endemic focus, are liable to infection. On account of the almost invariable tendency to undulations of pyrexial intensity Malta fever is often called "undulating fever," a name proposed by Hughes. The disease is also known as Gibraltar fever, Mediterranean fever, rock fever, etc., depending upon the locality.

The following is a list of places from which Malta fever has been reported:

Spain—Gibraltar; Islands of the Mediterranean—Balearic Islands, Corsica, Sardinia, Sicily, Malta, Gozo, Cyprus, Crete; Italy—Rome, Naples, Caserta, Benevento, Campobosso, Aricca, Terano, Fermo, Padua, Cittanova, etc.; Greece—Athens, Cephalonia; Turkey—Constantinople, Smyrna; Palestine—Jerusalem; Africa—Tunis, Algiers, Alexandria, Suakin, Massowah, Zanzibar, Kimberley (?), Aden; India—Calcutta, Mian-Mir, Nowshera, Secunderabad, Simla, Delhi, Lucknow, Agra, Allahabad, Choabattia, Subatha, Assam, Swat Valley; China—Hongkong; Philippine Islands; Fiji Islands; North America—Mississippi Valley (?); West Indies (?)—Cuba (?), Porto Rico (?); South America—Venezuela, Brazil, Montevideo.

Malta fever is a general infection not unlike other specific bacteremias, such as typhoid fever. The *Micrococcus melitensis* is found especially in the spleen and also in the blood. The inoculation of pure cultures of this organism into monkeys produces a prolonged febrile disease similar to Malta fever. There have been several instances of the inoculation of pure cultures into man, both intentionally and accidentally, which were followed by the characteristic symptoms of the fever after an incubation period of from five to fifteen days. Little doubt, therefore, remains that the organism is the true cause of the disease.

From the standpoint of prophylaxis it is of the first importance to determine the channel of infection by which the micrococcus enters the body. In the cases before mentioned in which the disease was produced by inoculating pure cultures of the *Micrococcus melitensis* into man, in one instance the culture was accidentally introduced into the conjunctival sac; in the others, by subcutaneous inoculation. One case which arose in England is supposed to have been conveyed from son to father by using a clinical thermometer in the mouth immediately after its use by the patient. From experimental evidence, therefore, it would appear that the infection of Malta fever may be taken in through wounds, the mucous membranes, or by food and drink introduced into the mouth. There is no evidence that the disease is directly contagious from the sick to the well.

Malta fever occurs especially in the officers and men of the British army and navy stationed at Malta and Gibraltar. All authorities recognize the influence of unfavorable hygienic conditions as an etiological factor of the greatest importance in prophylaxis. Sex has no predisposing influence and every age is prone to attacks, but it occurs mostly between the ages of 6 and 30 years.

In Malta the greatest incidence of the disease is in the hot, dry month of July. Chilling of the surface, bodily and mental depression, etc., are quoted as incidental causes.

The morbid process is that of a general infection and is seen especially in the condition of the spleen, which is enlarged, soft, even diffluent. The blood gives the usual picture of secondary anemia. The lymphoid elements are but slightly involved; the liver is congested and the seat of cloudy swelling, and the kidneys are sometimes swollen and show glomerular nephritis.

The period of incubation appears to be from a few days to thirty days, usually about fifteen.

On account of the large number of cases of Malta fever in the military and naval population of the island of Malta a commission was appointed by the admiralty, the war office, and the civil government of Malta in 1904 for the purpose of studying this disease with a view especially of determining the source of infection. This commission has issued six reports. These reports include a minute study of the general sanitary conditions of the island of Malta, the prevalence of the disease there, the various experiments upon the viability of the organism under many conditions, and experimental work upon susceptible animals. The following data in regard to the relation of goat's milk to the spread of Malta fever are largely drawn from these reports and, in many instances, are taken verbatim from the reports.

Until the researches of the commission the means of infection were not definitely known. Various theories had been suggested, such as the agency of biting insects, the ingestion of infected food and drink, the breathing of infected dust, and contacts.

Epidemiological studies having shown that, while the consumption of infected milk may and probably does account for Malta fever among the Maltese, yet many cases occur among the military and naval population in Malta which can not be attributed to this cause. Accordingly a study of mosquitoes as possible carriers of the *M. melitensis* was begun.

The *M. melitensis* was recovered four times from a total of 896 mosquitoes dissected. Deducting from these 896 mosquitoes those collected where there was no case of Malta fever or where the cases were mostly chronic we would have 4 infected mosquitoes out of 150 collected in presumably infected places. This result was not unexpected considering the small numbers of the specific organisms found in the peripheral blood of Malta fever patients. The mosquitoes could not be infected in great numbers or the disease would be much more prevalent than it is at present.

Captain Kennedy[a] was able experimentally to infect a monkey as the result of bites of mosquitoes (*Culex pipiens*) which had fed on patients suffering from Malta fever. An attempt to infect a monkey by bites from artificially infected mosquitoes, however, failed.

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[a] Reports of the commission * * * for the investigation of Mediterranean fever * * *. Part 4, 1906, p. 187.

In the examination of 103 cases of Malta fever for the specific organism the minimum quantity of blood from which a positive result was obtained was $\frac{1}{250}$ cubic centimeters. This fact has an important bearing on the question of the possibility of the transmission of infection by biting insects such as mosquitoes. This is a larger amount of blood than any biting insect to be found in Malta can contain.[a]

The water supply of Malta is drawn from two sources, the one for general use being derived from three springs which are pumped to a central reservoir and thence distributed, the second being rain water, most of the houses being provided with cisterns for the collection of rain water which is largely used for drinking purposes.

The milk supply of Malta is derived almost entirely from goats, though there is a small number of cows on the island and condensed milk is used to some extent. The number of milk goats in Malta is probably at least 20,000.

As showing the prevalence of the disease in Malta the following figures are of interest: From 1894 to 1903 there was an average of 32 cases per 10,000 inhabitants per year in the civil population; for the same period in the military population the yearly average was 25.6 per 10,000; from 1901 to 1903, for which years only figures are obtainable, the yearly average was 28.55 per 10,000 among the naval population.

In regard to infection other than through goat's milk, Major Horrocks[b] concludes that so far as the experiments go it appears that infection can not be conveyed from infected to healthy monkeys by skin contact alone, all other sources of infection being excluded. Infection can not be conveyed from infected to healthy monkeys by ecto parasites alone. When healthy monkeys living in intimate contact with diseased monkeys, under mosquito-proof conditions, become infected, the infection is due to the absorption of the *M. melitensis* excreted in the urine of the diseased monkeys.

There is no evidence that Mediterranean fever can be contracted by contact with cutaneous surfaces uncontaminated by urine.[c]

Infection can be acquired by the absorption of urine secreted by cases of Mediterranean fever, and this is probably one way in which workers in hospitals become infected.

There is evidence to show that monkeys can be infected by dry dust artificially contaminated with cultures of *M. melitensis* isolated from the spleen of cases of Mediterranean fever. The path of absorption may be through the nares, throat, respiratory passages, and alimentary canal. Dry dust contaminated with the urine of cases of Mediterranean fever has given rise to infection in goats, but not in monkeys. The experience gained during the work performed in Malta during 1904–5 has convinced Horrocks that men are more susceptible than monkeys and goats. Shaw's work on ambulatory cases of Mediterranean fever among the Maltese has also shown that opportunities for the creation of infected dust are plentiful in Malta. Infected dry dust as a cause of Mediterranean fever can not therefore be discarded. When infection is acquired in this manner the incubation period is probably at least a month.

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[a] Reports of the commission for the investigation of Mediterranean fever . Part 3, 1905, p. 11.

[b] Reports of the commission for the investigation of Mediterranean fever . Part 4, 1906, p. 36.

[c] Reports of the commission for the investigation of Mediterranean fever . Part 4, 1906, p. 81.

Mediterranean fever can be acquired by the absorption of infected goat's milk from the alimentary canal. The incubation period in this case is also probably long, and may even extend to two months.

This mode of infection probably plays a great part in the causation of Mediterranean fever among the Maltese, who drink raw milk drawn at the doors of their houses.

Horrocks[a] found that the *M. melitensis* could be recovered from khaki cotton, khaki serge, and blankets up to the eightieth day. Shaw recovered it from blue serge up to the seventy-eighth day.

The above results obtained by Horrocks upon the longevity of the organism upon khaki, cotton, etc., are important as showing the possible relation of fomites to the transmission of the disease.

The presence of ambulatory cases of Malta fever must be taken into account in the spread and continuance of the disease in Malta. These ambulatory cases constantly pass the specific organism in their urine and are undoubtedly as much a source of danger to those with whom they come in contact as are the bacillus carriers in typhoid fever.

The usual source of milk in Malta is the goat.[b] These animals are driven about the streets and milked at the customer's door into his own container. The udders, which are abnormally large, often touch the ground and are very liable to be soiled. There are so many herds that it is often difficult for a householder to tell the source of his milk supply. No regulations are in force for the effectual control of these vendors.

It was first shown by Zammit[c] that goats could be infected by feeding them with the *M. melitensis*. Zammit informed the chairman of the board that he considered goats to be susceptible to Malta fever and that the disease is spread to human beings by goats.

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[a] Reports of the commission * * * for the investigation of Mediterranean fever * * *. Part 4, 1906, p. 176.

[b] Reports of the commission * * * for the investigation of Mediterranean fever * * *. Part 2, 1905, p. 11.

[c] Reports of the commission * * * for the investigation of Mediterranean fever * * *. Part 3, 1905, p. 2.

On June 23, 1905, Maj. W. H. Horrocks wrote the chairman of the commission that he had discovered the *M. melitensis* in the milk of an apparently healthy goat and that he had already found it in the milk of five goats taken from two different herds, and that Doctor Zammit had found it in the blood of one of these goats.

Preliminary notes by Major Horrocks, Captain Kennedy, and Doctor Zammit on the propagation of Malta fever by goats show that one or more healthy goats in every herd are excreting the *M. melitensis* in their milk and urine, and that about 50 per cent of the goats react to Malta fever when examined by serum agglutination tests. The commission states that it may be objected that no exact proof exists that the drinking of milk containing the *M. melitensis* will give rise to the disease in man. However, when we take into consideration the results of feeding and inoculation experiments on monkeys it may be assumed that the disease is propagated in this way.

This is the first statement in the literature bearing upon the propagation of Malta fever by the milk of infected goats.

With the object of ascertaining by experimental inoculation whether goats could be infected by *M. melitensis* 6 goats from 2 different herds were brought and placed in the lazaretto. Doctor Zammit[1] before inoculation of these goats took blood from each and tested their serum for agglutination. He found to his surprise that the serum of 5 of these goats considerably diluted caused agglutination of the *M. melitensis*. The reactions thus obtained suggested that possibly 5 of the goats were suffering from Malta fever acquired under natural conditions. The goats were said to be healthy, but were sold cheaply as they had given very little milk for some time. Examination of these goats in detail resulted as follows:

Goat No. 6: *M. melitensis* appears to be steadily excreted in the apparently normal milk of this goat.

Goat No. 1: *M. melitensis* excreted in large number in the milk and also in the urine of this goat.

Goat No. 2: *M. melitensis* excreted in small quantities in the normal appearing milk of this goat; not detected in the urine.

Goat No. 3: *M. melitensis* present in large numbers in the normal looking milk of this goat, but not in the urine.

Goat No. 5: *M. melitensis* was found in the milk and urine.

Captain Kennedy, R. A. M. C., visited the various herds and took blood from the ears of the goats. Out of 161 goats examined 84 gave a positive agglutination test, equal to a percentage of 52 probably infected with Mediterranean fever.

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[1] Report of the commission * * * for the investigation of Mediterranean fever * * *. Part 1, 1905, p. 84 *et seq.*

The results obtained show that some of the goats in every herd examined were suffering from Mediterranean fever. The *M. melitensis* is present in the milk in enormous numbers when the disease has been present sufficiently long to cause a change in the physical characters of the fluid. It is also excreted in considerable numbers, even when the animals are in "full milk" and no changes have occurred in either the physical or chemical characters of the milk.

The *M. melitensis* is also excreted in the urine of goats suffering from Mediterranean fever, but up to the present it has only been found when the disease has existed for some time and after physical changes have occurred in the milk.

Shaw examined the blood of 33 cows, 10 of which gave a positive reaction to the *M. melitensis;* from the milk of 2 of these cows the *M. melitensis* was isolated.

The manner in which animals become infected with the virus of Mediterranean fever is a matter of considerable interest and importance. Up to the present all the evidence available points to their food as being the main vehicle of infection. The feeding experiments show conclusively that monkeys and goats may thus be infected. Besides the very obvious way of infection of the young through their mothers' milk, the successful result of various feeding experiments with food soiled, directly and indirectly, with the urine of 2 ambulatory cases of Mediterranean fever, and in whose urine living *M. melitensis* was being excreted, indicated another way in which these animals may be infected while feeding. Goats may be seen any day in the streets of the chief city of the island of Malta feeding on filth and rubbish of every possible variety, some of it visibly saturated with urine, animal and human. Among the lower class Maltese, as above stated, workmen have been found who void living *M. melitensis* in their urine, as do a certain number of infected goats. Thus the path of this manner of infection becomes clear. Having satisfied their hunger in this manner, the goats lie down in the streets to digest their meal with their teats and udders often in contact with the ordure of the gutters and roads, till they are kicked up by the goatherd to be milked into the vessel brought to the doors of the adjacent houses by their occupants. It is hence not to be wondered at that these animals frequently suffer also from suppurative mastitis and give milk containing pus. In the health reports of the Malta government may be seen reports of outbreaks of illness among children directly traced to this cause by the medical officers.

With regard to cows the evidence is not so clear. Kept shut up in "shippens," and seldom allowed outside, they have their food brought to them, but as this food is composed of vegetable and other refuse collected from every possible source and situation, it is easy to understand that they can hardly escape from receiving infected food from time to time.

It was interesting to note whether those goats whose blood gave a positive agglutination reaction would have some symptoms of illness, but this was not apparent except in a few cases. The quantity and quality of the milk seemed in most cases to be unaffected. In fact, it was often noted that the best milk-producers in the herd gave a positive reaction.

Horrocks and Kennedy[1] thought that as a result of their observations, judged by the serum reaction, 41 per cent of the goats in Malta are infected. Ten per cent of the goats supplying milk to various parts of Malta appear to excrete the *M. melitensis* in the milk.

The excretion of the specific microbe may continue steadily for three months without any change occurring in the physical character or chemical composition of the milk and without the animal exhibiting any signs of ill health. Some infected goats may lose flesh and their coats become thin: they may also suffer from a short hacking cough. A febrile condition, however, has not been observed. Goats may have a marked blood reaction and yet never excrete the *M. melitensis* in the milk. If the blood serum or milk does not agglutinate the *M. melitensis*, the specific microbe is not found in the milk.

The excretion of the *M. melitensis* in the milk may be intermittent, appearing for a few days and then disappearing for a week or more. A blood reaction may exist for some weeks before the *M. melitensis* is excreted in the milk.

Monkeys and goats can be infected by feeding with cultures of *M. melitensis* isolated from milk, and also by feeding with infected milk itself. The incubation period in feeding experiments appears to vary between three and four weeks. Monkeys infected by feeding sometimes suffer from a typical wave of fever and lose flesh, at other times they show no obvious signs of ill health, and may even gain in weight.

When monkeys become infected by feeding with milk the lymphatic glands always contain far more colonies of the *M. melitensis* than the spleen. This fact suggests that the specific micrococci contained in the food are carried to the lymphatic glands and there undergo considerable multiplication. It has not yet been proved that the mesenteric glands are always infected at an earlier date than the femoral and axillary glands, but feeding with milk shows that this may be the case at times.

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[1] Reports of the commission [illegible] for the investigation of Mediterranean fever [illegible] Part 4, 1906, p. 68, et seq.

It has been demonstrated that goats may become infected by feeding on dust polluted with urine from cases of Mediterranean fever. The excretion of *M. melitensis* in the milk resulting from such infection is a late phenomenon only appearing about seventy-four days after the blood reaction has developed.

In report No. 6 of the commission they state that, reviewing the evidence already collected by the Mediterranean Fever Commission in its entirety, it is fairly obvious that the infective character of the milk of many of the goats upon the island of Malta affords a ready and reasonable explanation of the means by which the disease is transmitted. Then, too, the evidence yielded by experiments upon monkeys, supported by the facts of the steamship *Joshua Nicholson* epidemic, justifies the assumption that in the ingestion of infected milk we have the veritable infective agency in the vast majority of cases. Additional weight attaches to this view by reason of the declining case incidence that was associated with the compulsory substitution (owing to the goatherds strike) of imported preserved milks for the fresh goat's milk by the local and military authorities.

In report No. 6, page 70, is an account of an outbreak of Malta fever aboard the steamship *Joshua Nicholson* which conveyed a herd of milk goats from Malta to Antwerp in the latter part of the summer of 1905. These goats were collected by a representative of the United States Bureau of Animal Industry for shipment to the United States. It reads almost as if it were a planned laboratory experiment, and in view of the experimental work above referred to established almost conclusively the relation of infected goat's milk to the spread of Malta fever. The following account of the outbreak is taken verbatim from the report of the commission:

1. HISTORY OF THE GOATS.

Mr. Thompson, of the United States Bureau of Animal Industry, visited Malta in the summer of 1905, and during a stay of some months gradually purchased a herd of 61 milch goats (all healthy in appearance and good milkers, many being prize animals), and 4 billy goats. These he shipped on board the cargo steamer *Joshua Nicholson*, on August 19, 1905, for passage to the United States via Antwerp. During the voyage, which lasted until September 2, 1906, when Antwerp was reached, the goats were milking well, and many of the ship's company partook freely of the milk—the officers drinking "mixed" milk collected in a large vessel, the members of the crew each obtaining "whole" milk from 1 goat in his own separate panikin.

On arrival at Antwerp the goats were at once transferred to the quarantine station, where they remained for the five days that elapsed before they were reembarked on the steamship *St. Andrew* bound for New York, and during this voyage a large quantity of milk was again available for consumption. New York was reached about September 24, and the animals were transferred to the quarantine station at Athenia, N. J., where they remained under observation. Subsequent bacteriological examination resulted in the recovery of *M. melitensis* first from the milk of 2 of the goats and afterwards from that of several more.

2. THE INCIDENCE OF MEDITERRANEAN FEVER AMONG THOSE WHO PARTOOK OF THE MILK.

(*a*) *In the steamship Joshua Nicholson.*—In addition to 4 passengers (Mr. Thompson and 3 goatherds) present on the voyage from Malta to Antwerp, the *Joshua Nicholson* carried 23 officers and men. Of the crew of 19, the carpenter, boatswain, and messroom steward, together with others (11 in all), left the ship at Antwerp; the boatswain was afterwards in hospital suffering from hernia; the movements of the remainder can not be traced. Of the 12 remaining officers and crew, 8 fell sick at intervals varying from eighteen to thirty-four days from the embarkation of the goats, and in the cases of 5 of these 8 the blood reactions leave no room for doubt that Mediterranean fever was the cause of their illness.

The 4 members of the ship's crew who did not show any signs of illness were the second mate and the cabin boy, with whom the milk disagreed and who consequently had but very little, and 2 engineers (Germans) who drank the milk, it is true, but appear to have always boiled it.

Of the 3 goatherds, 1 (the chief goatherd) had undoubtedly been infected with *M. melitensis* previous to July, 1906, as evidenced by the presence of specific agglutinins in his blood, but whether recently or remotely it was impossible to say; about the 2 assistant goatherds no information could be obtained.

(*b*) *At Antwerp.* The staff of the quarantine station and many individuals in the neighborhood are said to have partaken of the milk, both raw and boiled, during the five days the goats were interned here, but no information can be obtained of the subsequent occurrence of cases of illness resembling Mediterranean fever.

(*c*) *In the steamship St. Andrew.*—The steamship *St. Andrew* carried 30 cattlemen and 3 goatherds, and Mr. Thompson, in addition to a crew of 30 men. Most of these drank of the milk, but the master of the ship and also his owners state that none of the men suffered from any illness.

(*d*) *In America.* With the exception of Mr. Thompson, who died in January, 1906, from "bilateral pneumonia following influenza," and about whose medical history, qua Mediterranean fever, no evidence can be obtained, only 1 person—a woman at the quarantine station—took the milk in any quantity. She, however, drank the mixed milk from several goats for a considerable period, and in December, 1905, suffered from a typical attack of Mediterranean fever.

3. THE RESULTS.

In summarizing the result of this unpremeditated experiment several factors have to be considered. For instance, a certain unknown number of goats—more, however, than 2—were shown to be secreting infective milk after their arrival in America, some three months after leaving Malta, but there is no direct evidence as to the number whose milk contained *M. melitensis* during the voyage in summer weather from Malta to Antwerp. Arguing from analogy with average Maltese herds, at least 6 should have been secreting infective milk. The goats purchased by Mr. Thompson were, however, picked animals and heavy milkers, and as experience has shown that the goats yielding the most milk in any given herd are the most likely to be passing *M. melitensis* in their milk, the probability is that in this particular herd of 60 milch goats (1 having died the day after leaving Malta) the milk from considerably more than 6 was heavily infected—an inference which receives confirmation from the fact that the 3 officers and the steward who drank "mixed" milk each developed an attack of Medditerranean fever, the remaining officer and the cabin boy, with whom the milk disagreed and who consequently did not drink it, remained well.

The members of the crew, on the other hand, each drank "whole" milk from a single goat, and apart from the possibilities of the milk being supplied on any particular occasion from an uninfected animal, a reference to Section I (3), shows clearly the possibilities of a man who obtains milk, even from an infected animal, avoiding the ingestion of infective milk.

Apart from such considerations, however, it suffices to state the net result as follows:

Of 23[a] men on board the steamship *Joshua Nicholson* who drank on one or more occasions presumably infected milk, no evidence whatever is available as to 12 and no relevant information as to Mr. Thompson; of the remaining 10, 1 suffered from hernia only, 1 was infected by *M. melitensis* at an unknown date, while 8 suffered from febrile attacks 5 (or 50 per cent of them) yielding conclusive evidence of infection by *M. melitensis.*

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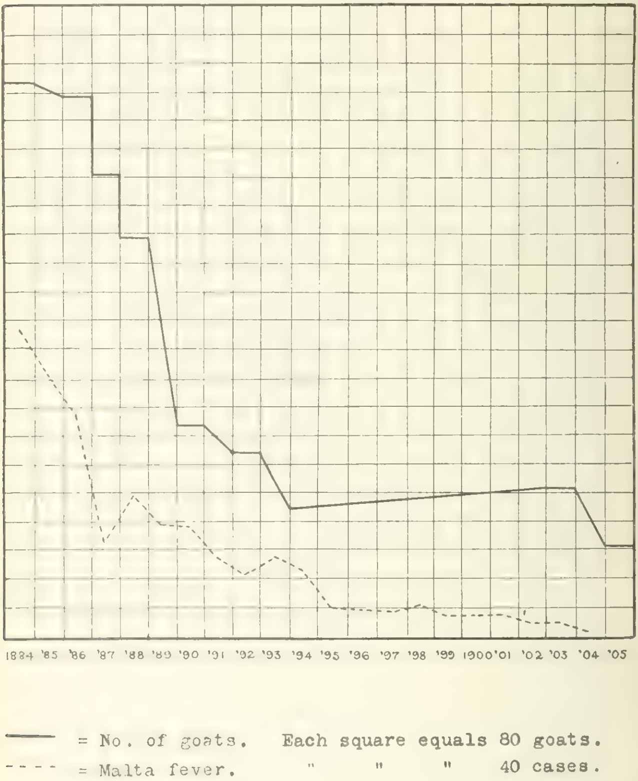
[a] That is disregarding the 2 men who boiled the milk before drinking it, and the officer and cabin boy who did not drink the milk.

In Report No. 5 of the commission is an article by Major Horroks on Mediterranean fever in Gibraltar. The facts there detailed, taken with the curve showing the relation of the number of goats in



Gibraltar to the number of cases of Malta fever, is particularly interesting and suggestive. With the reduction in the number of goats in Gibraltar there was also a decrease in the number of cases of Malta fever, so that finally when the number of goats had decreased to about 200 in 1905, Malta fever has practically disappeared.

Mediterranean fever, often called "rock fever," has existed in Gibraltar for many years,[a] although the cause of the fever was not known until the discovery of Bruce's specific organism from fatal cases of Malta fever. Physicians residing in Gibraltar knew of the existence of a fever characterized by long duration, low mortality, and liable to be followed by rheumatic sequela.

In a study of the cases of continued fever in Gibraltar from 1882 to 1905 it was shown by Horrocks that in the year 1884 there were 833 cases of continued fever of which 429 were probably Mediterranean. In 1885 there were 697 cases of continued fever including 341 cases of Malta fever. In 1886 there were only 331 cases of continued fever and of these 158 were enteric fever. The great increase in the number of cases of enteric fever was attributed partly to the arrival of an infected regiment in Gibraltar from Egypt and to serious sanitary defects in Gibraltar. In 1887 there was again a considerable falling off in the number of cases of Mediterranean fever and from that date, with slight oscillations, the curve of Mediterranean fever gradually declined until it reached 0 in 1904. The rapid disappearance of febrile diseases from Gibraltar, which commenced in 1885, forms a marked contrast with the state of things in Malta in corresponding years. It is plain that some important factor which disappeared from Gibraltar has continued to operate in Malta.

It has been shown that the *M. melitensis* is excreted in the urine of man and goats and that animals can be infected by dust contaminated with the urine of Malta fever patients; that the micrococcus is excreted in the milk of infected goats, and that the consumption of this milk causes Malta fever in monkeys. It is evident that both the sanitary conditions and the possible infection of goats in Gibraltar must be investigated if the cause of the continued fever is to be discovered.

That the improvement of the sanitary conditions played but a minor part in the marked decrease in the prevalence of Malta fever in Gibraltar is shown by the fact that the curve representing the presence of Malta fever among the military population rose steadily from 1874 to 1884, in spite of the improvement in the sanitary conditions in Gibraltar.

Twenty years ago goats were allowed to graze on certain portions of the rock and passes were granted to goat keepers for this privilege. In 1883 passes for 1,793 goats were granted. In 1886 the number of passes had been reduced to 1,512; by 1890 the passes had further declined to 590, and in 1892 to only 510. From 1894 to 1902 the number of goats appears to have changed very little; in 1901 the passes were reduced to 210, and when Major Horrocks began an examination of the goats in 1905 he found only 254 distributed upon various portions of the rock. It might be urged that, though passes for grazing were withdrawn, the goats were still kept and housed in goat sheds. This, however, was not the case, as Major Horrocks ascertained that in the period from 1883 to 1903 about 1,100 goats had been sold, and those familiar with the goat trade stated that where passes for grazing could not be obtained the goats were not kept in any numbers.

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[a] Reports of the commission * * * for the investigation of Mediterranean fever * * * Part 5, 1907, p. 55.

In 1905, Major Horrocks took specimens of blood from 254 goats found on various portions of the rock and tested the serum for agglutination. Fourteen per cent of them gave a positive reaction with *M. melitensis*. It is extremely suggestive that the decrease in the Malta fever in the military population was coincident with the decrease in the number of goats in Gibraltar.

It appears probable that the rapid disappearance of Mediterranean fever from Gibraltar, which commenced in 1885, was intimately associated with the exodus of infected goats from the rock. Improved sanitary conditions, especially the disconnection of waste pipes and house drains from sewers, may have played a part in causing the decrease of fever, but as the same sanitary improvements have been carried out in Malta without any corresponding decline of Mediterranean fever, it is fair to assume that their effect was insignificant compared with that produced by the removal of the infected goats.

6. MILK SICKNESS.

MILK SICKNESS.

By George W. McCoy,

Passed Assistant Surgeon, Public Health and Marine-Hospital Service.

Definition.—Milk sickness is an acute, nonfebrile disease, probably of a specific nature due to the ingestion of milk, milk products, or the flesh of animals (usually cattle) suffering from a disease known as trembles. The disease in man is characterized by great depression, persistent vomiting, obstinate constipation, and high mortality.

Synonyms.—Endemic sick stomach, sloes or slows, milk sick, sick stomach, colica trementia, puking complaint, paralysis intestinalis, mukosma, syro.

Historical.—Milk sickness was first noted and its association with trembles in cattle first defined about the beginning of the last century. The earliest professional account appears to have been published by Drake in 1809, and was based upon the observations of Dr. Thomas Barbee. Since that time the disease has become an important part of the medical history of our middle west.

In some localities the disease was so prevalent and fatal that whole communities migrated from "milk-sick" sections to parts where the disease did not occur.

Almost every community in some parts of the country has a tradition about outbreaks of this disease in the earlier years of the past century. We are told by Colonel Henry Watterson (1909) that Nancy Hanks, the mother of Abraham Lincoln, died from the disease in 1818 after an illness of a week. In the words of Colonel Watterson, "The dread milk sickness stalked abroad, smiting equally human beings and cattle."

With the advance of civilization, as forests were cleared and pastures fenced, the disease became less frequent; by the time of the civil war the disease was by no means common. At the present time it is one of the rarest of diseases. Trembles in animals is now almost as rare as "milk sickness" in man. According to Jordan and Harris (1908) an active focus of the disease exists in the valley of the Pecos River in New Mexico, where the disorder among animals has generally been attributed to "alkali poisoning." An outbreak occurred in Macon County, Tenn., in April and May, 1907. Small epi demies are reported in some part of Tennessee every two or three years. The cases now occur only in the thinly settled regions, usually remote from lines of communication. Most frequently they are attended by a layman, known locally as a "milk-sick" doctor, who has a local reputation for curing the disease. The only modern scientific contribution to the literature of milk sickness is the work of Jordan and Harris.

Milk sickness in man (and trembles in animals) was such an important question in the early years of the last century that several State legislatures offered liberal rewards for the discovery of the cause of the disease. In 1821 the legislature of Tennessee passed an act requiring fences to be made around certain coves in Franklin County "to prevent animals from eating an unknown vegetable, thereby imparting to their milk and flesh qualities highly deleterious."

At the present time when the disease is rare many persons living in and near the endemic foci abstain from the use of milk and butter on account of the danger of contracting milk sickness.

At the outset one is confronted with the difficulty that the affection under consideration is largely a matter of tradition. Satisfactory accounts of the disease are rare. Drake (1841), who is much quoted in all accounts of the disease, appears not to have been personally familiar with the malady; indeed, in his memoir he states that he has seen no case in man nor in the lower animals. Yandell (1852), who is also frequently quoted and has written much on the subject, makes no mention of having himself seen cases, and in his later publications expresses grave doubt as to the existence of a specific disease corresponding to that described as milk sickness. In his own words: "Upon a review of the whole matter, the conclusion to which all the testimony on the subject has brought me is, that we, who have written upon milk sickness have been egregiously imposed upon by careless and incompetent observers." Many of the accounts, indeed I think I may safely say, the majority, are based upon hearsay evidence.

A large number of the articles published on milk sickness were written wholly with the object of proving that a plant poison is the cause of the disease; many others, that a mineral poison is the causative agent. The disease has been described as a mild, almost trivial affair; and again as most malignant and fatal. A few writers have regarded the disease as a manifestation of malarial poisoning. However, the mass of the testimony clearly indicates that there is a specific disease, known as milk sickness, always derived from a case of trembles in an animal.

Distribution. So far as known the disease has never occurred outside of the United States. In this country it has been endemic in many of the newly settled regions, in practically all of the States south of New York, and as far west as Missouri and Arkansas. Tennessee, Kentucky, Ohio, Indiana, and Illinois have suffered the most severely. At the present time cases occasionally occur in Tennessee and North Carolina. Cases have been reported in Illinois as late as 1904. The recent discovery of the disease in New Mexico is the first indication we have of its occurrence west of the Mississippi Valley.

In the endemic foci, the disease in the lower animals is limited to rather well-defined areas. Many of these areas are fenced to prevent the access and consequent contamination of stock. I have seen a number of such inclosures varying in size from an acre to several thousand acres. "Milksick Mountain," in White County, Tenn., is entirely inclosed by a fence 7 or 8 miles in length, built about fifty years ago; since which time the disease has been very rare in that locality.

The infected areas are always wooded land, but otherwise vary markedly, from dark damp ravines to high dry ridges or ordinary level forest tracts. Seaton (1841), who wrote extensively on the subject, claimed that the disease was found only where sandstone entered largely into the composition of the soil. Other writers do not agree with this view.

There appears to be a very general agreement in the opinion that wooded land is essential for the existence of the disease and the clearing of the land suffices to remove all danger of animals acquiring trembles. It is said that if land be rendered harmless by clearing, then be permitted to produce a new growth of timber, the tract may again become the seat of the disease. So sharply are some milk sick areas defined that farmers point out places where on one side of a fence animals may be pastured in perfect safety, whereas if pastured on the other side of the fence they are almost sure to contract trembles. I have been told of more than one outbreak of trembles due to changing the fence of a pasture by a few yards so as to include some wild (uncleared) land.

It has been claimed that springs and water courses have conveyed the cause of trembles, but it seems clear that in such cases the animals contract the disease in the surrounding wooded areas.

Etiology and pathology. Children appear to be less liable to the disease than adults. Nursing women are said to enjoy a relative immunity (Johnson, 1866). One attack confers no immunity; in fact, it appears to predispose to subsequent attacks (Philips, 1877).

The disease occurs most frequently in the spring and the fall, but records of cases in summer are not rare and a few are said to have occurred in winter. Drake (1841), who investigated the subject in Ohio, states that the disease occurred in May and June, but was more frequently met with in August, September, October, and November. The majority of writers agree with this, stating that cases are most frequent in the fall months, and especially when the season has been dry. The last outbreak in Tennessee occurred in April, 1907, and the general impression prevails among physicians and laymen in that State that the disease occurs only in the spring and the autumn.

So far as milk sickness in man is concerned about the only etiological fact of importance is that the disease occurs as a result of the use of milk, butter, cheese, or flesh from an animal suffering from trembles. Even this has been questioned. Yandell (1867) states "that the relation of the disease to animal products is not on an impregnable basis." The great mass of evidence, however, leaves little doubt but that the disease is practically always derived from a case of trembles.

The favorite theory among physicians and laymen is that trembles is caused by a poisonous plant eaten by the animals. It is supposed that the poison is eliminated in the milk, or if the animal is not in lactation is stored up in its tissues. In support of this theory it is urged that the disease occurs only in seasons when animals are allowed to graze in the open, and only when they graze in certain special places that soon become known as milk sick. A number of plants, notably poison ivy, white snakeroot, and certain mushrooms, have been claimed to be the essential cause of the disease. These plants are all common in many localities that have never had milk sickness, and in no case does the claim that any one of them is the cause of the disease appear to be well founded. Indeed the flora of a milk-sick region may be identical with that of the adjacent healthy land.

Next in popularity to the plant poison theory of the cause of the disease is the mineral-poison theory.

Seaton (1841) very vigorously maintained that milk sickness was a form of arsenic poisoning. Lead and cobalt have also been accused.

I have been unable to bring about any condition in guinea pigs that even remotely resembles trembles by feeding experiments with cobalt, lead, or arsenic. When these animals finally succumbed to the poison their tissues were without any harmful effect on animals (guinea pigs) to which they were fed.

Two facts, apparently well established, may be urged against either the plant or mineral poison theory. In the first place, the flesh of animals dead of either trembles or milk sickness will, when eaten by another animal, cause that animal to develop trembles and the disease may again be reproduced by feeding the flesh of the second animal. It is said that this transference of infection may thus be carried through a long series of animals. In the second place, the observation has been made very frequently that, under natural conditions, it is only exposure at night or in the morning while dew is on the grass, that is capable of infecting an animal with trembles.

The limitation of trembles to certain well defined areas, and the fact that night exposure only appears to be dangerous, suggest the possibility of the conveyance of the infection through an intermediary host, such as arthropods or biting insects.

A favorite theory many years ago was that the disease is produced by a gas or miasm rising from the earth in the affected region. The gas was supposed to be generated by earth or vegetation. At the present day, no discussion of this theory is necessary.

Trembles was early recognized to have some of the features of infectious diseases. In 1843, Heeringen wrote "I am compelled to believe that trembles belongs to the anthrax family." This was twelve years before the discovery of the anthrax bacillus. Mention is frequently made of the fact that the disease may be carried from one animal to another by feeding the flesh of a diseased animal.

In 1877, Philips reports finding "spiral bacteria" in the blood of a typical case, and the same organism, with cocci, in the urine of the same case. He encountered similar organisms in the urine of other cases.

Gardner (1880) reported finding in the blood of a heifer suffering from the trembles, organisms "that bore in size and behavior a striking resemblance to the form of bacteria called by naturalists bacilla subtilissima." He found the same organism in the water of a spring that had supplied a family in which milk sickness was present. Dogs suffering from "slows" acquired by eating the flesh of the heifer also had the organism in the blood. He also found the organism in milk.

Graff (1841) reported some very remarkable experimental work with trembles. He found the flesh of the animals not to differ materially in appearance from that of sound animals. Salting meat, he says, does not impair its poisonous properties. The milk of a cow was poisonous, as shown by feeding it to dogs for eight days after she was removed from the infected pasture; but a test made a week later showed the milk to be harmless. He found small amounts of meat or butter sufficient to cause the disease. "One ounce of butter or cheese or 4 ounces of beef, either raw or boiled, administered three times a day, will certainly prove fatal within six days, and often earlier." All these experiments were upon dogs and the flesh of his experimentally killed dogs was as poisonous as the beef that conveyed the disease.

Graff found that treating the flesh with dilute sulphuric acid for two hours did not destroy the poison; even heating had no effect. He says butter heated "to such a degree as to cause it to inflame lost none of its poisonous properties." He failed to extract the poisonous agent from meat by prolonged boiling. He failed in attempts to communicate the disease "by an inoculation with any portion of the body or secretions from infected animals." These experiments lack confirmation.

Jordan and Harris have isolated a micro-organism from the tissues and body fluids of animals suffering from trembles, which they have called "*B. lactimorbi.*" The following is a brief abstract of their description of the bacterium:

The organism is a motile rod, and appropriate staining demonstrates the presence of flagella. Spores are found under certain conditions. On an agar slant the growth is smooth, grayish, glossy, without pigment formation. There is a turbidity of broth at the end of twenty-four hours, and later a pellicle forms, which falls when the tube is agitated. Litmus milk is at first rendered alkaline, later it turns dirty-white, and finally may become opalescent. No multiplication occurred on potato. On Löfflers blood serum there is a smooth, yellowish growth. Gelatin is slowly liquefied.

The nonsporulating cultures are killed by an exposure of five minutes to a temperature of 55° C., while the spore-bearing cultures are destroyed at 100° C. maintained for fifteen minutes. The disease has been reproduced in a rabbit by the inoculation of blood from an infected animal. Feeding experiments have shown that the dog and the calf may be infected with the organism, which may in turn be recovered from their tissues after death. These observers report having isolated the organism from several naturally infected cows and from one naturally infected horse, and Doctor Jordan informs me in a personal communication that they have also isolated it from a man and from sheep.

It would appear from this work that another of the diseases, the cause of which has long been shrouded in doubt and mystery, has at last yielded its secret to laboratory investigation.

Milk cows seldom show any symptoms so long as they are regularly milked, even though they are secreting milk fatal to man and to other animals; in a herd the steers and heifers always show symptoms before the cows that are giving milk. Buttermilk is generally regarded as harmless. Graff thought differently, however.

Apparently not all are equally susceptible, as it has frequently been noted that of several persons who partake of the poisonous milk or meat, some may escape, while others, usually the majority, will contract the disease.

A recent outbreak which I have investigated had some of the conditions of an experiment on human beings. The record, unfortunately, is based entirely upon nonprofessional observation, but is, I believe, fairly accurate. In brief, it is as follows: Seven persons partook of a meal, 6 of whom used milk and butter and became ill with characteristic symptoms of milk sickness and subsequently died. The only person who escaped was a woman who never used either milk or butter. One of the 6 was a guest and had only this one meal in this house. This individual sickened on the day after partaking of that meal. The other 5 persons became ill at different times; the last one about ten days after eating the meal that apparently poisoned the guest. A calf using the same milk sickened with "trembles" soon after the earliest cases in the family. The cow accused of imparting the disease developed "trembles" and died. The cow showed no symptoms until milking was neglected on account of illness in the family. It was believed that this cow had been on milk-sick land about two weeks prior to the outbreak. This outbreak seems to have been a typical one, the sickening of the cow only after she was no longer milked, the sickening of the calf at about the same time that some of the persons were attacked, the onset of the illness at a varying period after the use of the suspected milk and butter, finally, the exemption of the only person who did not partake of the milk or butter, all agree with the older descriptions. As trembles and milk sickness are both so rare at present, an occurrence like this points strongly to a most intimate relation between them.

The few recorded post-mortem examinations throw little light upon the nature of the disease. Horne (1844), who examined three human cases, found inflamed patches in the small intestine. The mesenteric glands were red and greatly enlarged.

In animals, Graff found the brain "suffused with a large quantity of blood, which, from the amount contained within the cranium, must have made great pressure on every part." In one human case he found softening of the brain and evidence of meningitis. Graff tells us that this autopsy was conducted "by stealth at night in the open air, and by the light of a single candle."

Barbee (1840) found the colon in man "contracted to the size of a common candle." The mucous membrane of the stomach was red and thickened in spots; the remainder presented a pale and softened appearance. The peritoneal coat of the small intestines was inflamed.

Jordan and Harris made a number of post-mortem examinations on the lower animals. They noted the odor of acetone when the body cavities were opened. This is interesting in view of the statement of most of the old writers that there is a peculiar and characteristic odor of the breath in milk sickness. The other findings in cattle were, briefly, as follows:

Small amounts of fluid in pleural and pericardial sacs, numerous ecchymoses beneath the visceral pericardium. The heart muscle was paler than normal and when sections were examined general cloudy swelling was found. A general injection of the vessels of the small intestine was present. The liver was always enlarged, purple red, sometimes with streaks of yellowish. Microscopical examination showed cloudy swelling and fatty degeneration of the organ. The gall bladder was usually full of dark-green bile. The liver tissue was very friable, occasionally yellowish red, and gave the appearance of the "nutmeg" liver. The spleen and the kidneys were markedly congested. The mucous membrane of the small intestine was deeply injected and had much tenacious mucus adhering to it. In horses the lesions were similar to those described in cattle. The liver showed marked cloudy swelling and less fatty change than in cattle. There were small nodules embedded in the wall of the small intestine. These nodules were 4 or 5 millimeters in diameter and were elevated above the surface. They were found to originate in the lymph nodes embedded in the mucosa.

Symptoms.—Philips (1877) and others thought that an interval of days or even weeks elapsed between the exposure on infected areas and the development of symptoms of trembles in cattle. Drake describes the symptoms of trembles in animals as follows:

> The animal begins to mope and droop, and to walk slower than its fellows, to falter in its gait. If under these circumstances it should be driven, and attempt to run, the debility and stiffness of its muscles are immediately apparent. It fails rapidly, trembles, pants, and sometimes seems blind, as it runs against obstacles, but this may arise from vertigo; at length it falls down, lies on its side quivering, and is not, perhaps, able to rise for several hours, sometimes never.

He also mentions a chronic form.

The characteristic symptom, trembling, may always be brought out by exercising the suspected animal. It is related that cattle buyers never purchased animals from milk-sick districts until they had given them a run of half a mile or more to ascertain if they had trembles.

When a cow is regularly milked no symptoms are likely to develop.

In at least some instances a period of several days appears to intervene between the consumption of the poisoned milk or meat and the onset of symptoms in man. Spalding (1881) reports an outbreak where three days in one case and six days in another intervened between suspending the use of the suspected milk and the onset of the symptoms. He also speaks of the onset in some cases as being "almost instantaneous when milk or beef is taken." It would appear that such cases, with very early onset, may be due to decomposition products belonging to the class of poisons usually called ptomaines.

As judged by the description of most writers, the symptom complex in man appears to be fairly uniform. In describing it I will use freely the account of Way (1893). The onset is gradual, the individual tires easily, there is loss of appetite, in a day or two vomiting begins, the bowels are obstinately constipated, there is great abdominal distress, the tongue becomes large and flabby, the breath acquires a foul odor that is regarded as highly characteristic of the disease, the abdomen is scaphoid, there is marked visible pulsation of the abdominal aorta, the temperature is not elevated; in fact, it is generally subnormal, there is always great thirst. The mind usually remains clear, but in fatal cases, coma for several hours may precede dissolution. The average duration of cases is about one week. The cases referred to in the recent outbreak in Tennessee died in from two to ten days after the onset of symptoms.

A common sequel of milk sickness is a lasting debility. I have seen a considerable number of persons who claimed that since an attack of the disease, they were incapacitated for hard work, especially in warm weather.

The mortality is quite high. Physicians who have had a large experience with this disease tell me that at least half the cases will perish, even when carefully treated. Numerous family outbreaks are recorded where the mortality has been 100 per cent, as was the case in the last outbreak in Tennessee.

Treatment.—The early settlers had worked out the very successful preventive treatment of keeping their animals from lands known to be dangerous, or what is better, to use for purposes of pasture in endemic foci, only "tame" lands; that is, land from which the timber had been cut. It is even better to bring the land under cultivation, but this does not appear to be essential.

With our present knowledge the treatment of the disease should be purely symptomatic. We have no specific remedy. Rest in bed, abstinence from food, stimulating enemeta, and a judicious use of stimulants would appear to be indicated.

The treatment of cases in the early days was somewhat vigorous in accordance with the therapeutic customs of the day. Graff recommended free drawing of blood and the use of calomel not to exceed 5 grains every two or three hours. Some advised a much more liberal use of calomel. Counter irritation over the abdomen was a favorite measure used to allay abdominal pain and vomiting. It was generally regarded as essential to secure a free movement of the bowels, and when this had been accomplished the case was regarded as offering a favorable prognosis.

Drake (1841) considered blood letting of doubtful value, but advised the free use of cathartics. Enemeta were frequently used. Philips (1877.) used a purely expectant plan of treatment and urged against the use of strong purgatives. He used strychnine in liberal doses, apparently with benefit.

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7. THE RELATION OF COW'S MILK TO THE ZOOPARASITIC DISEASES OF MAN.

THE RELATION OF COW'S MILK TO THE ZOO-PARASITIC DISEASES OF MAN.

By Ch. Wardell Stiles, Ph. D.,

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Summary.—Theoretically, it is possible that certain infections with animal parasites may be contracted through the milk supply, but such possibility does not present any danger which is even remotely comparable with the danger of contracting typhoid through the milk. No animal parasite is known for which milk is a necessary transmitting medium or a necessary habitat in any particular stage of the life cycle. Accordingly, the danger of contracting zoo-parasitic diseases through the milk supply is in general more theoretical than real, and can be prevented by the most elementary methods of cleanliness.

There is no animal parasite known for man for which cow's milk is either the necessary medium of transmission or the necessary habitat during any portion of its life cycle. The question of the relation of cow's milk to the zoo-parasitic diseases of man reduces itself therefore to the question as to what animal parasites of man are most likely to gain access to the milk accidentally during a stage of their life cycle which would render their transmission to man possible.

In reference to this question the broad statement may be made that such possible cases would in general be due to the following causes:

(*a*) Fraudulent practices on the part of persons in the milk trade in diluting the milk with water.

(*b*) The use of contaminated water either in such cases or in washing the utensils with which the milk comes into contact.

(*c*) Improper disposal of fecal matter.

(*d*) Careless personal habits on the part of milk dealers, servants, etc., whereby the milk might, by coming into contact with their hands, become infected with stages in the life cycle of the parasites which would render transmission possible.

(*e*) Carelessness whereby fecal material from various animals (particularly of dogs, rats, and mice) might gain access to the milk; and

(*f*) Permitting cats or dogs to have access to the milk or to the dishes used for milk.

From the foregoing it will be seen that the entire question under discussion is one of simple, elementary cleanliness, honesty, and propriety; that when due regard is had for these three factors the danger of infection by animal parasites, through the milk supply, is eliminated; but that such danger increases in proportion as these factors are ignored.

There is no evidence on record that any one of the foregoing possibilities has ever played an important rôle in producing any large number of cases of infection. Still it may be well worth while to refer to these possibilities briefly as contributing arguments in favor of a clean milk supply.

(*a and b*) *Water-borne parasites.*—If contaminated water is used in washing milk cans or in fraudulently diluting milk it stands to reason that the contamination in question may be transmitted to the milk and through the latter it may be transmitted to the consumer. In this manner any obligatory or faculative water-borne zoo-parasitic infection (such as amebic dysentery, coccidiosis, possibly some forms of distomatosis, cysticercosis, hydatid diseases, eelworms, etc.), might be transmitted through the milk. The dangers involved are not sufficient to cause any sensation or alarm, but they are sufficiently real to present contributing arguments in favor of protecting milk from foul and contaminated water.

(*c*) *Improper disposal of fecal material.*—When fecal material is not properly disposed of, the danger is present that the infection which it contains may be spread in various ways, as by flies, to the food, and thus it may gain access to man. The danger involved in reference to the animal parasites is not, in general, so great as it is in reference to the bacterial infections—such as typhoid, cholera, etc.; for in case of the zooparasites the transmission in most of the instances in which it is theoretically possible could take place only when the organisms had reached a certain stage in their life cycle. For instance, a typhoid or a cholera stool would, a priori, be more dangerous when fresh than when one to several weeks old, and its danger would decrease with age; from a case of amebic dysentery, hookworm disease, or eelworm infection, danger from a perfectly fresh stool would in general be nil; gradually the stool would become infectious corresponding to the rapidity of the development of the infecting stage of the parasites in question; this infectiousness would increase to a maximum, according to conditions of heat and moisture, and then the infectivity would gradually decrease. If stools in an infective condition are visited by flies or are washed into a water supply or are scattered in dust form, they can, according to the various conditions, form the basis of various zoo-parasitic infections, and should particles of such stools be accidentally carried to milk, the milk could act as a mechanical bearer of the germs. In general, however, the chances of such method of infection seem rather remote, in so far as the animal parasites are concerned.

(*d*) *Personal habits of persons who handle milk.*—It seems possible that the personal habits of persons (such as milkers, servants, etc.), who come into more or less close contact with milk, might be a more appreciable element than any of the foregoing in infecting the milk, although even in such cases the infection in question, namely, by animal parasites, would be of far less importance than a typhoid infection. For instance, while a milker who is a typhoid "bacillus carrier" would be an element of grave danger to the public health, an infection (in that person) of pinworm, of pork tapeworm, and perhaps of Cochin China diarrhea, might be of some slight importance, in reference to the possibility of their transmission through the milk supply; but if that person had coccidiosis, the fat, or the broad tapeworm, or flukes, eelworms, or whipworms, such infections would be without significance, so far as the public milk supply is concerned.

(*e*) *Fecal material from animals.*—It is not a pleasant thought that our milk supply may contain fecal material from various animals but such is unfortunately the case. Upon several occasions other divisions in this laboratory have submitted to the Zoological division for determination, sediment taken from bottled milk and such sediment has proved to be feces from rodents—either rats or mice. Now it is supposed that at least 3 intestinal parasites from the rats and mice are capable of developing directly in man. In the case of one of these parasites (dwarf tapeworms), the usually accepted view is open to question, since the form in man is perhaps specifically distinct from the form in rodents; in the case of a second parasite (the trichina worm), the transmission from rat's feces to man is probably possible, but more theoretical than practical; in the case of a certain protozoan infection (*Lamblia*) it is quite possible that a real, though perhaps not very frequent danger is present of its transmission through the milk supply.

(*f*) *Infections from dogs and cats.*—Probably the greatest danger of the transmission of parasites from dogs and cats through the milk supply lies in the accidental infection with hydatids, from contamination with canine feces, and the accidental presence, in milk, of the cat and dog flea, in which a larval tapeworm occurs which is transmissible to man. In neither case, however, is any instance of these parasites positively traced, so far as I know, to this method of infection, although such method must be admitted as theoretically possible.

8. MORBIDITY AND MORTALITY STATISTICS AS INFLUENCED BY MILK.

MORBIDITY AND MORTALITY STATISTICS AS INFLUENCED BY MILK.

By J. M. Eager,

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The influence of milk on morbidity and mortality furnishes a striking example of the potency for evil of a thing designed for the accomplishment of good. The food of the new-born and the most important aliment of the sick and the aged becomes too often a promoter of disease and an instrument of death. This malign influence of impure milk or milk improperly used is made evident by the mournful proofs of the extensive and growing statistics on the subject.

QUANTITIES OF MILK CONSUMED.

The importance of the rôle played by milk in the causation of disease is emphasized when attention is drawn to the enormous quantities of milk consumed. Based on the Twelfth Census of the United States taken in the year 1900, the milk and cream sold in 1899 by farmers, deducting the quantities purchased by butter and cheese factories and condensed-milk establishments, was equivalent to about 740,000,000 gallons of milk. This quantity of milk consumed by the nonfarming population in a single year was as great as the quantity of water supplied to the city of Washington in about ten days. The average quantity of milk purchased by the urban and suburban population of the United States is 23 gallons a year for each person. The consumption of milk in Philadelphia during the year 1905 was estimated at 23 gallons for each inhabitant; and in London, England, during the year 1892, at 11.5 gallons.

MILK AND DISEASE.

Health may be influenced by cow's milk either because the milk is physiologically unsuitable, as for infant feeding, or because it has become a medium of infection. Milk of inferior nutritive value can not be without its effect on the health of the consumer, especially when used as a food for babes. This effect is difficult to show statistically.

Its potency is nevertheless evident from a statement by J. Wicliffe Peck, chemist to the Hospital for Sick Children, London, that the average quality of milk offered for sale throughout London is so defective in fat and nonfatty solids that a child at six months, whose weight should increase about 4 ounces weekly, suffers each week a loss in diet of 3½ ounces of fat and nonfatty solids when its ration of fraudulently manipulated cow's milk is based on the supposition that the milk is of standard quality. Such low standard milk tends to produce marasmus and rickets. Marasmic children present a decided predisposition to bronchitis and summer diarrhea and thus indirectly an increase of infant mortality is brought about by diluted or adulterated milk.

STATISTICS OF INFANTILE MORTALITY.

The malign effects of hand feeding of infants and the consequent impress made upon the mortality returns become manifest by an examination of vital statistics, but to gauge the exact ratio of infant deaths resulting from artificial feeding is very difficult.

The reports of the United States Census Office on mortality for the year 1905 show that in the registration area with a population of 33,757,811 there were, of 545,533 deaths at all ages from all causes, 105,553 deaths among infants under one year of age. Diarrhea and enteritis caused the death of 39,399 infants in the first year of their life. In England and Wales all the deaths registered during the same year numbered 520,031 and were in the proportion of 15.2 per 1,000 persons living. The deaths of infants under one year of age were in the proportion of 128 per 1,000 births in the year as compared with 150 per 1,000, the mean proportion in the years 1895 to 1904, inclusive. The proportion of infant deaths in England and Wales during the year 1905 is the lowest then recorded, although closely approximated in some previous years.

Commenting upon the official statistics of infantile mortality the Registrar-General of England writes:

> It has frequently been pointed out in the reports that although the general mortality in this country has steadily fallen in the course of the last half century, nevertheless infants in the first year of life have not shared in the benefit. About one fifth part of the total loss of life in the first year after birth takes place within a week of that event, while by the end of the first month the proportion reaches one third, and by the end of the third month it exceeds one half. From the first to the fourth month diarrheal diseases steadily increase in destructiveness, after which month they become gradually less fatal, although they still contribute seriously to the death rate throughout the first year of age.

The rate given for the whole of England and Wales does not fairly represent the infant mortality of the cities of England. It was stated, for example, at the annual meeting in 1906 of the subscribers to the

Children's Hospital at Pendleberry that the death rate of infants under one year during the decade ending 1903 was 183.8 in Manchester and 198.3 in Salford.

The following statistics from various countries are along the same lines:

Country.	General mortality—Deaths to 1,000 living; average annual rate in 10 years (1895–1904).	Country.	Infantile mortality—Deaths of children under 1 year to 1,000 births—average annual Rate in 10 years (1895–1904).
Russia (European)	[a]33.6	Chile	[b]326
Chile	28.8	Russia (European)	[a]268
Spain	27.8	Austria	[c]224
Hungary	27.3	Roumania	[d]218
Ceylon	26.8	Hungary	216
Roumania	26.8	Prussia	197
Austria	25.2	Spain	[e]182
Servia	23.9	Jamaica	176
Bulgaria	23.9	Italy	170
Italy	22.7	Ceylon	169
Jamaica	22.4	Belgium	156
German Empire	20.8	France	153
Prussia	20.5	Servia	154
Japan	[b]20.5	Japan	[a]151
France	20.4	England and Wales	150
Finland	18.7	The Netherlands	147
Switzerland	18.1	Western Australia	147
Ireland	18.0	Bulgaria	[b]144
Belgium	17.8	Switzerland	142
Scotland	17.8	Finland	134
England and Wales	17.2	Denmark	127
The Netherlands	17.0	Scotland	126
Denmark	15.8	New South Wales	108
Sweden	15.8	Victoria	105
Norway	15.1	Ireland	103
Western Australia	14.6	South Australia	102
Victoria	13.3	Queensland	101
Tasmania	11.8	Sweden	[b]98
Queensland	11.8	Tasmania	94
New South Wales	11.7	Norway	90
South Australia	11.5	New Zealand	79
New Zealand	9.8		

[a] Average for 10 years (1892–1901).
[b] Average for 10 years (1894–1903).
[c] Average for 8 years (1895–1902).
[d] Average for 10 years (1890–1899).
[e] Average for 5 years (1900–1904).

Balestre and St. Joseph, in a study of mortality in early infancy in the urban population of France from 1892 to 1897, give a mass of valuable data bearing on the appalling annual toll exacted from the infant population of France—a loss of life which in conjunction with the unusually low birth rate in France has given the question of infantile mortality in that country a national importance.

Infantile mortality in France.

[1892 to 1897, inclusive.]

Place.	Deaths at all ages from all causes.	Deaths of infants under 1 year per 1,000 of all deaths at all ages.	Deaths from diarrhea and gastro enteritis of infants under 1 year per 1,000 deaths under 1 year from all causes.
Paris, population 2,511,629	303,206	145.35	380.30
11 cities between 100,000 and 500,000 inhabitants having a combined population of 2,365,238	322,129	184.73	420.49
47 cities between 30,000 and 100,000 inhabitants having a combined population of 2,421,820	334,032	167.25	350.06
622 cities of less than 30,000 inhabitants having a combined population of 5,892,034	790,576	168.13	(a)

[a] Not recorded.

In Paris and the cities of France having over 30,000 inhabitants the deaths from diarrhea by months per 1,000 infants under 1 year dying from all causes were as follows:

January	212.8	July	587.1
February	211.1	August	606.4
March	224.8	September	537.7
April	254.8	October	431.5
May	303.1	November	304.6
June	426.4	December	235.9

It is seen from these figures that, though the months of June, July, August, September, and October present the most deplorable proportion of deaths from diarrhea, this cause is not negligible in autumn and winter.

In Germany, according to Behring, of every 1,000 children born alive 235 succumb during the first year of life. Only 510 out of 1,000 males born attain manhood. Not more than a third of those reaching maturity are found to be fit for military service. These sad facts Behring attributes very largely to the ulterior effects of infection derived in infancy from milk.

DIARRHEAL DISEASE AND MILK.

The statistics given show how large a proportion of all deaths are among infants. It has been said that there is only one other period in life in which the chance of death is greater than it is under 1 year, namely, in persons over 90 years old. It is seen also that no cause is so prolific among children in the first year of life as disease of the digestive organs. Diarrheal diseases of infants are generally accepted to be due to impure food; but there is no reason to believe that the alimentary canal of the average infant is often incapable of digesting the food necessary for growth and development when the food supplied is of suitable quality and quantity. During the first year of its life a child consumes about 500 quarts of milk. There is ample evidence to show that the proportion of deaths among infants is greatly reduced when they receive the food nature designed for them, namely mother's milk, or when, as a substitute therefor, the most exact imitation is provided with due care to prevent its infection. There is no doubt that the nursing of all infants by healthy mothers would contribute immensely to the reduction of the infantile death rate. Observations in many parts of the world confirm this conclusion.

MOTHER'S MILK AND LESSENED INFANTILE MORTALITY.

Casper in 1825 recorded that a trustworthy traveler, von Schubert, says that the high death rate among young children in Norway and Northern Sweden in the early part of the last century was very evidently due to feeding infants with cow's milk instead of mother's milk. At present, breast nursing is altogether the custom throughout Norway, insomuch that Borchart quoting statistics in 1883 says that in Norway and Scotland where suckling of infants is the rule, out of 100 children born 10.4 for Norway and 11.9 for Scotland die, whereas in Würtemberg, where mothers as a rule are not in the habit of suckling their infants, 35.4 per cent perish in the first year of life. W. J. Tyson states that of all infants who die in England in the first year of life three-fourths have been fed artificially, and Doctor Hope, medical officer of health of Liverpool, says that according to his observation sanitary conditions have no marked influence on infant mortality, but that the methods of infant feeding are chiefly responsible for the high rate at which it is maintained.

Newsholme, with a view to determining the relation of mortality to artificial feeding, gives a census of an infant population of 1,259 in 10,308 houses in Brighton, England, taken in a house to-house inspection in the three years 1903–5, inclusive, combined with an inquiry into the manner of feeding of 121 babes dying of diarrhea and belonging to the same social stratum as those forming the sample population. He concludes from these inquiries that, taking the whole of the first year of life, the number of deaths from epidemic diarrhea among breast-fed babes is not much more than one-tenth the number among artificially fed infants. Considering separately infants aged from 6 to 9 months, bearing in mind the fact that breast fed babes at this age must have been breast-fed from birth, he finds that 57 per cent of such babes were entirely and an additional 17 per cent partly breast-fed. Not one of the deaths at the age in question occurred among breast-fed or partially breast-fed children.

By a similar inquiry the results obtained at Brighton were confirmed in the borough of Finsbury, England, by an investigation by

Sandilands in which the method of feeding of 695 infants was investigated. It was ascertained that of 139 infants under 9 months of age dying of diarrhea 16 per cent only were breast fed. Of the survivors, 69 per cent were breast-fed.

France furnishes facts of the same import. In 1898, when diarrhea made many victims among the children of Paris, it was estimated that the number of deaths of artificially fed infants was double that of the breast fed at all times during the year and that in August it ran up to 8 times that of the breast fed.

Before a deputation, in 1906, of the Queensland government on the subject of infant life protection, Turner reported that during the summer months at Brisbane, Australia, more than one-half of the bottle-fed babes die.

INFANTILE MORTALITY A CLASS MORTALITY.

Harrington points out that infantile mortality is a class mortality, highest as a rule in cities and towns where women work in industrial establishments and put their children early to the bottle. In an article written in 1906 he gives a table prepared from the United States census in which mill towns in New England are shown to have the greatest infantile mortality.

Reid, as a result of a careful inquiry made in 1906, shows that the infantile mortality rates are in great excess in the northern artisan towns of Staffordshire, England, where pottery is the chief industry and women, both married and single, are engaged in factory labor. This excess is very marked where a comparison is made with the southern towns of the county where mining and iron-working prevail, affording practically no employment for women. The general conditions which operate in causing a high infantile mortality prevail, it is pointed out, to an equal extent in the two populations. The difference in the death rate among infants in the two sections is attributed to the nature of the trades as affecting the employment of women away from home, with the consequent effect on the proportion of breast fed and bottle-fed infants. The percentage of female married and widowed factory workers to the whole female population between the ages of 15 and 50 years was studied in different localities. In 5 towns in which the percentage of such women so employed was 12 or more the infantile mortality was 198 per 1,000; in 13 towns in which the percentage was under 12 and over 6 the infantile mortality was 156, and in 8 towns in which the percentage was under 6 the infantile mortality was 149.

While the relation between factory labor for women and the death rate of young children seems well established for Staffordshire the statistics of 1,000 towns given by the Registrar-General of England for 1905 do not show such a relation throughout England to be intimate.

UNNECESSARY HAND FEEDING.

Although it might seem useless to repeat what the greatest medical authorities have so often asserted it is interesting nevertheless to consider to what extent hand-feeding, with its melancholy impress on vital statistics, is an absolute necessity. Madame Dluski, in a thesis delivered at the Baudelocque Clinic, Paris, expresses the opinion that among 100 healthy women, when the necessary conditions of alimentation and repose are present, 99 are actually able to nurse their offspring. She concludes that women, almost without exception, can nurse their babes; that four-fifths of mothers can do so from the beginning of lactation; that nearly all can do so after a longer or shorter time, and that absolute agalactia does not exist. Yet despite all efforts to promote the practice of breast-feeding a great proportion of infants are uselessly bottle fed. Indeed the practice of feeding infants with the milk of animals (goats and cows) is of great antiquity—the Greeks and Scythians had recourse to it—but it is believed to be greatly increasing in modern times.

SCIENTIFIC ARTIFICIAL FEEDING AND THE MORTALITY RATE.

In consequence of the great diffusion of the practice of artificial feeding for infants it is interesting to study the effect on morbidity and mortality statistics of a scientifically compounded artificial diet compared with a diet too often ignorantly or carelessly prepared. The statistics of the pasteurization of milk throw much light on the subject.

THE STRAUS PASTEURIZED MILK DEPOTS.

Pasteurized milk was first made available for infants in general in New York City in 1893, in which year Nathan Straus dispensed 34,400 bottles of milk so prepared from one depot. In 1894 339,194 bottles were issued, in 1895 666,622, and in 1896 666,911. In 1905 2,668,397 bottles were dispensed and 1,016,731 glasses of pasteurized milk were bought at the booths in the parks of New York City. In 1906 17 Straus stations dispensed 3,142,252 bottles of pasteurized milk and 1,078,405 glasses.

Prior to the beginning of this work the death rate of children under 5 years in New York City was over 96.2 out of every 1,000 and in June, July, and August the death rate of children was at the rate of 136.4 per 1,000 per annum. With the increased use of pasteurized milk the death rate fell to 55 per 1,000 in 1906, and the summer death rate to 62.7 per 1,000.

These figures, year by year, are given in the following table, compiled from the official statistics of the New York Department of Health:

Population, deaths, and death rate of children under 5 years.

Year.	Population.	Deaths.	Death rate per 1,000 per annum.
1891	188,703	18,224	96.5
1892	194,214	18,684	96.2
1893	199,885	17,865	89.3
1894	205,723	17,558	85.3
1895	212,983	18,221	85.5
1896	217,071	16,907	77.9
1897	221,339	15,395	69.6
1898	225,804	15,591	69.1
1899	230,480	14,391	62.5
1900	235,585	15,648	66.4
1901	242,747	14,809	61.0
1902	250,153	15,019	60.0
1903	257,813	14,402	53.3
1904	265,738	16,137	60.7
1905	273,938	15,287	55.8
1906	282,423	15,534	55.0
FOR THE MONTHS OF JUNE, JULY, AND AUGUST.			
1891	188,703	5,945	126.4
1892	194,214	6,612	136.1
1893	199,886	5,892	117.0
1894	205,723	5,788	112.6
1895	212,983	6,183	116.1
1896	217,071	5,671	104.5
1897	221,339	5,401	97.6
1898	225,804	5,047	89.4
1899	230,480	4,689	81.4
1900	235,585	4,562	77.4
1901	242,747	4,642	76.5
1902	250,153	4,389	70.2
1903	257,813	4,037	62.6
1904	265,738	4,805	72.3
1905	273,938	4,892	71.4
1906	282,423	4,426	62.7

For the purpose of comparison, the figures are confined to the original city of New York now the Boroughs of Manhattan and the Bronx.

At the rate of mortality of 1892, there would have been 27,169 deaths of children under 5 years in 1906, instead of 15,534. Thus the apparent saving in one year was 11,635 lives, or 42.82 per cent. At the summer mortality rate of 1892, death would have claimed 9,743 victims in June, July, and August, 1906, instead of 4,426. Thus the apparent saving of lives in three months was 5,317, or 54.57 per cent.

It must not be overlooked that in New York City, coincidently with the introduction of pasteurized milk, other agencies became operative, as for example general milk inspection by the local health authorities, the use of diphtheria antitoxin, the campaign of fresh air for children, improved tenement houses, cleaner streets, more parks and playgrounds, recreation piers, and other factors accompanying the enlightenment in hygiene so widely spread in recent years, not only in New York City, but throughout the whole country.

STATISTICS OF RANDALL'S ISLAND.

When the infants in the care of the city of New York were fed on milk from a carefully selected herd pastured on the island, the death rate was as follows:

	Children treated.	Number of deaths.	Percentage.
1895	1,216	511	42.02
1896	1,212	474	39.11
1897	1,181	524	44.36
Total	3,609	1,509	41.81

A pasteurizing plant was installed in the early part of 1898. No other change in diet or hygiene was made.

	Children treated.	Number of deaths.	Percentage
1898	1,284	255	19.80
1899	1,097	269	21.52
1900	1,084	300	27.68
1901	1,028	186	18.09
1902	820	181	22.07
1903	542	101	18.63
1904	345	57	16.52
Total	6,200	1,349	21.75

Had the ratio of deaths for the three years, 1895, 1896, and 1897, been maintained in the seven years from 1898 to 1904, the total infant mortality would have been 2,592, instead of 1,349, a difference of 1,243.

STATISTICS OF MILK CHARITIES ABROAD.

Writing of infantile mortality and the supply of humanized sterilized milk, Hope states that at the Liverpool infant milk depots for three years ending with the year 1903, among 4,153 infants provided with the depot milk the mortality rate was 78 per 1,000, compared with the following infantile mortality rate in the city of Liverpool:

Year.	Infantile mortality.
1901	188
1902	163
1903	152
Average	167.6

Harris has prepared the following comparative table showing results of the St. Helens depot in the town of St. Helens, England:

Year.	Number of children on the books.	Death rate per 1,000 among children at depot.	Infantile death rate, borough of St. Helens.
1899	232	103	157
1900	332	102	188
1901	282	106	175
1902	200	82	167

Lederer states that as a result of the system of pasteurization in practice in Vienna for the past seven years the proportion between the mortality rates for breast-fed and bottle-fed children which formerly was 1 to 20 is in latter years between 1 to 5 and 1 to 8. It is observed that in Vienna the improvement in artificial diet reduces the mortality in the second year of life also.

In France there are two types of organizations, the *Consultation de Lait* and the *Goutte de Lait*, having for their object the encouragement of breast feeding wherever possible and a supply of properly prepared milk to those infants for whom breast feeding is impracticable. At the *Consultation de Nourissons* of the Clinique Tarnier, Paris, the annual mortality rate during a period of about six years among 712 children who attended the *Consultation* from birth for an average period of nine and one-half months was 46 per 1,000. Reference for comparative purposes to the death returns of Paris during the years 1898, 1899, and 1900, shows that there was a mortality of 178 per 1,000 among infants under 1 year of age.

MILK AS A DIET FOR THE SICK.

The influence of impure milk on the duration of sickness and on the death rate when milk is employed as an invalid diet is difficult to demonstrate statistically. For the sick, milk—usually uncooked milk—is often a principal or an exclusive article of diet. Considering the increased susceptibility of feeble and aged persons to infection and the diminished resistance offered by the sick, there can be no doubt that contamination of milk is a factor that plays a part in keeping up the rate of sickness and death.

MILK AND TUBERCULOSIS.

The report of the United States Census Office on mortality for the year 1905 shows that deaths from all causes in the registration area were in the proportion of 1,616 per 100,000. Tuberculosis in all its forms caused 193.6 deaths per 100,000. *Applying the same rate throughout the United States, it may be justly estimated that tuberculosis causes over 160,000 deaths a year in the United States.*

At the International Congress on Tuberculosis held in London in 1901, Koch made the announcement that bovine tuberculosis is transmissible to the human subject to only a slight extent if at all. The doubt thus cast on the relation between cow's milk and tuberculosis has to a great extent disappeared on further investigation made by a host of observers, most prominent among whom is von Behring, who claims that milk fed to infants is the chief cause of tuberculosis in man.

Schroeder and Cotton in a recent bulletin of the Bureau of Animal Industry conclude that the assertion that tuberculosis is a negligible quantity in the measures that must be taken for the preservation of human health is without basis and that there is no more active agent than the tuberculous cow for the increase of tuberculosis among animals and its persistence among men.

The rarity of primary intestinal tuberculosis, on which subject there is a discrepancy of statistics, is not in favor of the theory of infection by ingestion. It has been, however, repeatedly proved that tubercle bacilli may pass through a mucous membrane without leaving traces at the point of entrance. Again it has been demonstrated by competent observers that tubercular infection may take place through the tonsils. Latham estimates that not less than 25 to 30 per cent of the cases of tuberculosis which occur in early childhood are due to intestinal, and therefore presumably to food, infection. Of deaths in 1905 from all forms of tuberculosis in the registration area of the United States, about 1 in 39 was among infants under 1 year and 1 in about 14 among children under 5 years of age.

Ravenel writing in 1898 says:

> In northern Norway, Sweden, Lapland and Finland where reindeer contribute the bulk of farm animals, or about Hudson Bay and the islands of the Pacific, where there are only a few cattle, tuberculosis is far less prevalent in man. In Algiers the cattle are few and live for the most part in the open air and away from cities and it is found that tuberculosis does not increase among the natives. In Italy, on the other hand, where cattle are housed, Perroncito states that tuberculosis has become the scourge of man and beast.

Regarding the conveyance of tuberculosis in the colder countries, Cobb points out that an absence of tuberculosis does not necessarily follow the absence of milk from the dietary. He shows on trustworthy evidence that the Alaskan Indian, including the Esquimo and Aleut, is the victim of consumption of the lungs to a great and increasing extent, though these people do not use to any extent milk of any kind as an article of diet, and cow's milk not at all. Of interest in this connection is the report made in 1906 by the medical officer of health of the city of London showing that at least 8 per cent of the milk sold within the city limits of London is derived from animals affected with tuberculosis, and that of 500 cows examined after slaughter by the city veterinarian evidence of tuberculosis was found in 46.8 per cent.

EPIDEMICS CAUSED BY MILK.

In epidemics caused by milk (typhoid fever, scarlet fever, diphtheria, etc.), the mortality of the disease does not appear to differ from that of the same disease otherwise conveyed. The effect of milk epidemics on morbidity and mortality returns may be surmised by the frequency with which epidemics of such a character occur.

MILK AND TYPHOID FEVER.

Raudnitz, of Prague, states that one-fourth of the epidemics of typhoid fever in Austria are traceable to contaminated milk, and McCrae records that an inquiry into the causation of 638 epidemics of typhoid fever showed that in 17 per cent the infection was conveyed by milk. The bearing of this observation on the general sick and death rate is obvious when it is considered that the mortality in typhoid fever, though often as low as 5 per cent in private practice, sometimes reaches 20 per cent. Typhoid fever causes more deaths than any of the other epidemic diseases. The United States census reports show that in 1905 there were 28.1 deaths from typhoid fever per 100,000 population. The death rate from typhoid fever was smaller in 1905 than in any of the five preceding years. The annual average for the registration area of the United States, 1900 to 1904 inclusive, was higher than that for any of the countries given in the following table except Italy:

Deaths from typhoid fever per 100,000 of population.

Country.	Annual average, 1900 to 1904	Country.	Annual average, 1900 to 1904.
Registration area of United States	33.7	Sweden	12.2
England and Wales	12.9	Hungary	28.3
Scotland	12.7	Belgium	20.2
Ireland	14.2	Switzerland	6.5
Germany	8.5	Italy	37.8
Norway	6.2		

SCARLET FEVER AND DIPHTHERIA.

The number of epidemics of scarlet fever and diphtheria where the infection was conveyed by milk show unmistakably that the effect on morbidity and mortality rates thus brought about by milk must be considerable. While the death rate is low among patients of the better classes, in hospitals and among the poor it ranges from 5 to 30 per cent, a marked variability of the death rate in different epidemics being a characteristic of scarlet fever. The general mortality of scarlet fever is shown by the following table:

Number of deaths from scarlet fever per 100,000 population.

Country.	Annual average, 1900 to 1904	Country	Annual average, 1900 to 1904.
Registration area of United States....	11.8	Hungary	67.0
England and Wales	12.7	The Netherlands	2.6
Scotland	10.8	Belgium	16.3
Ireland	4.7	Switzerland	3.7
Germany	23.7	Spain	5.9
Norway	5.8	Italy	1.6
Sweden	8.7		

Diphtheria and croup caused an annual average of 33.6 deaths per 100,000 in the registration area of the United States, 1900 to 1904. Among the means of transmission of diphtheria infected milk is a well-recognized medium.

DISEASES OF CATTLE.

Numerous other diseases in the transmission of which milk is a factor exert an effect on vital statistics. Milk sickness, a disease related to the affection in cattle known as the trembles, still occurs in certain parts of the United States. It is transmitted by milk and milk products as well as the flesh of diseased animals. In some of the Western States in early days it was a prominent disease and killed many of its victims.

Foot and mouth disease in the cow has been frequently transmitted to human beings by the use of milk and milk derivatives. In one epidemic thus brought about the death rate was 8 per cent.

ASIATIC CHOLERA.

Milk is not infrequently a means of communicating Asiatic cholera. The evil efficacy of milk thus infected in its influence on morbidity and mortality statistics can be readily conjectured, when the desolating death record of cholera is reviewed and the almost universal use of milk considered.

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9. ICE CREAM.

ICE CREAM.

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The use of artificially frozen dishes as an article of diet is not of very ancient origin. It is not the purpose of this paper to discuss the physiological and dietetic effects of introducing ice-cold foods into the stomach. There are grave objections to the practice which will occur to every physiologist and hygienist. Briefly I may state that the process of digestion in the stomach depends upon the free excretion of the peptic ferments by the glands of the inner coats of the stomach. The introduction of large quantities of ice-cold material can not fail to contract the orifices of these glands and check their excretory activity.

Aside from this, however, the question of ice cream is one of grave importance in connection with the dairy supplies of the country, and particularly so because under the name of ice cream are found upon the markets products of the widest variation in composition, varying from the true ice cream to the true frozen pudding.

It is necessary, therefore, in the discussion of the matter, if possible, to ascertain first, what ice cream is or should be, and second, to study the materials from which it is made with a view to determining their sanitary character, and finally to determine the composition of the article itself as it is offered to the market. Incidentally therefore the dairy which furnishes the milk and the milk which furnishes the cream are subjects of inquiry. These two subjects, however, have been carefully gone over in other papers of this series and hence any reference to them will be merely of an incidental character as illustrating some point in connection with the particular subject at hand.

The term "ice cream" is used in this country to cover a large variety of products, which in Europe are known under the general term of "ices." The Neapolitan ices are said to be a type of the European dishes. This type of ices is found in most of the cities of Europe, served often in very attractive packages with various adornments or used directly without molding upon the table. The art of representing different kinds of fruits and flowers, animals, and other objects is also said to be of distinct European origin, although copied very largely in this country. For this reason there may be seen in both countries frozen products representing fruits of every description and usually colored and flavored to imitate the fruits which they represent. Strawberries, apples, pears, lemons, oranges, pineapples, peaches, apricots, bananas, grapes, and nearly all other fruits are thus represented. Various figures of statuary, or public buildings, or objects of art are also imitated in the form of frozen packages of this description. Even when milk or cream is used in the composition of these frozen dainties in Europe it is not the custom to call them ice cream. The Italian general name for these dishes is "sorbetto," the German is "Gefrorenes," the French "glacé," and the English "ice." With the exception of the frozen dish called "sherbet," practically all the forms known in Europe under the names given are called, or have been called until recently in this country, "ice cream."

In the discussion of this problem I shall first offer the investigations made under the auspices of the committee appointed by the District Commissioners to advise them in regard to the dairy products on sale in the District, including a study of the raw material from which ice cream is made and of the ice creams themselves. These studies have been conducted both from the chemical and bacteriological points of view.

I will afterwards give a brief historical sketch of the use of the term ice cream and the compounds to which it has been applied.

Next will be presented certain data respecting a proper standard for ice cream, a standard adopted by the United States Department of Agriculture under authority of an act of Congress, and the criticisms of this standard made by manufacturers and dealers in ice cream.

In this way it is believed that the whole subject may be presented in such form as to be useful not only to the Commissioners of the District in any work which they may inaugurate respecting the control of ice cream, but also to the people of the District and the people of the country in general.

It is not deemed advisable to go into minute details respecting the bacteriological and chemical investigations. I will content myself therefore with presenting the tables of analytical data and with giving a summary of the chemical and bacteriological investigations.

SUMMARY OF CHEMICAL DATA RELATING TO CREAM.

The samples of cream which were purchased in the open market covered a period extending from January 30, 1907, to June 12, 1907, inclusive. (See Table III, page 300.)

For the purpose of this investigation the analytical data reported referred only to the percentage of fat in the cream and to its artificial coloring. The analyses were made in the dairy laboratory by and under the supervision of Mr. G. E. Patrick.

The total number of samples examined is 132, including one double cream excluded from the averages. The average percentage of fat therein is 19.09.

By act of Congress the legal standard of fat in cream for the District of Columbia is 20 per cent. The number of samples at or above 20 per cent is 44, or 33.58 per cent. The number of samples below 20 per cent of fat is 87, or 66.41 per cent.

These data show that only one-third of the samples of cream purchased complied with the legal standard for the District. The standard for fat in cream, established by the Secretary of Agriculture under authority of Congress for the country at large in so far as interstate commerce is concerned is 18 per cent. The number of samples examined which are found at or above 18 per cent is 82, equivalent to 62.60 per cent of the total number. The number of samples below 18 per cent is 49, or 37.4 per cent of the whole number.

The data show that as sold upon the markets of Washington during the time mentioned almost two-thirds of the commercial creams complied with the national standard. The total number of samples of the above lot which are found to contain more than 25 per cent of fat is 6; the number of samples containing less than 16 per cent is 24; the number of samples containing less than 14 per cent is 6, and the number containing less than 13 per cent is 3, all of which are from the same dairy. These data show that the requirement of 18 per cent of fat, judged by the ordinary commercial data, is entirely just and satisfactory. Hence it follows that ice cream made from standard cream will easily contain 14 per cent or more of butter fat for the vanilla type of ice cream, and 12 or more per cent for the fruit type of ice cream, thus showing that the standards established are reasonable and just from the commercial conditions which actually exist. Of the total number of samples examined 15, equivalent to 11.45 per cent, are found to be artificially colored, thus showing that the artificial coloring of cream is not practiced to any great extent, and its entire prohibition would not in any way disturb the existing conditions of trade.

SUMMARY OF THE CHEMICAL DATA RELATING TO ICE CREAM.

The chemical analyses of the ice creams were made in the dairy laboratory of the Bureau of Chemistry by and under the supervision of Mr. G. E. Patrick, chief of that laboratory. (See Table IV, page 303.)

For the purpose of this report only the fat content of the various samples of ice cream, the presence of gelatin, vegetable thickeners, and coal-tar dyes are reported. The summary of the chemical data show the total number of samples analyzed to be 228. Judged by the standard of 14 per cent for the ice creams of the vanilla type and 12 per cent of fat for the ice creams of the fruit type, it is found that there are at or above standard 117 samples, or 51.32 per cent, and below standard 111 samples, or 48.68 per cent. The average percentage of fat in the entire 228 samples is 12.67. Only 46, or 20.18 per cent of the whole number of samples, contain less than 10 per cent of fat, and only 25, or 10.97 per cent, contain less than 8 per cent.

The total number of samples containing a thickener was 80, or 35.18 per cent. In 33 samples, or 14.47 per cent, the thickener is gelatin, while in 47 samples, or 20.61 per cent, the thickener is a vegetable gum or starch. Only 2 samples are found to contain coal-tar dye.

These samples were purchased at random from all the principal ice cream makers in Washington, over a period extending from about April 1 to August 1, 1907.

The data are most interesting in view of the contention that the standard suggested for butter fat is too high, and especially in view of the fact that 8 per cent has been suggested by many as a proper standard. The chemical examination shows how devoid of commercial significance are both of the claims mentioned. Another interesting fact is that the percentage of samples containing gelatin is extremely small. This is of great significance as being a most emphatic negative answer to the contention that gelatin is necessary to the manufacture of ice cream, or is generally employed. The chemical data on the whole give no support to the contention that the suggested standard for ice cream is unfair. The absence of eggs, gelatin, starch, and other substances, which it has been said are commonly used in the manufacture of ice cream, from the great majority of the samples is another point of great significance. In fact, the data show most conclusively that the term ice cream, even from a commercial point of view, is applied to a substance containing more than 14 per cent of fat in more than 51 per cent of all the samples examined. It is, therefore, commercially as well as scientifically and hygienically, a term which should be applied to a substance of standard composition and that standard, in so far as Washington is concerned, could be reached with but little variation from the usual methods of producing ice cream. What is true of Washington certainly should be true of other cities, since there is no indication that the quality of the creams made in Washington is any better than that of other cities.

The only conclusion which can be derived from the study of these chemical data is that the term ice cream should apply generally, as it does in the majority of cases at the present time as indicated by the results of these investigations, to a product made principally of cream and sugar, and with a natural flavor, either of an ordinary flavoring substance like vanilla or of fruit. Hence there appears to be no reason for departing from the established standard, in view of the data which have been secured by an examination of the commercial samples bought in the open market from all portions of the city.

BACTERIOLOGICAL INVESTIGATIONS OF ICE CREAM IN THE DISTRICT OF COLUMBIA.

[Made by or under the direction of Dr. George W. Stiles, and by or under the direction of Dr. M. E. Pennington.]

In most instances the samples of ice cream received for examination were collected directly from the original place of manufacture. In a few cases, however, miscellaneous samples were taken at places other than those at which the product was prepared. Generally half a pint or a 10-cent box furnished a sufficient quantity to make the chemical, microscopical, and bacteriological examinations. The 10-cent box, to which reference is made, was the pasteboard carton almost universally used as a container for ice cream when sold in small quantities, and for this reason it was much preferred as a carrier of the samples to be investigated. When these cartons were not available well-cleansed bottles or new paper boxes were used instead.

Upon arriving at the laboratory, samples for bacteriological examination were removed at once from the frozen interior by means of sterile spoons and placed in sterile dishes to melt. Generally within eight to ten minutes a sufficient liquefaction had occurred to enable the experimenter to remove enough material to make the bacteriological examination.

The enormous number of organisms which are found in cream, milk, and ice cream, necessitates high dilutions to make possible the quantitative determination of the organisms present. For the making of these, and the counting of the colonies which developed, the technique pursued may be stated briefly as follows: The quantities were measured in 1 cubic centimeter pipettes, graduated in 0.01 of a cubic centimeter, and 10 cubic centimeter pipettes graduated in 0.1 of a cubic centimeter. They were sterilized by heat and kept in bacteria-proof metal cases.

In order to make the necessary dilutions Erlenmeyer flasks of about 500 cubic centimeters and 100 cubic centimeters capacity, respectively, were used. To the former were added 99 cubic centimeters of sterile water and to the latter 9 cubic centimeters. To the flask containing 99 cubic centimeters there was added 1 cubic centimeter of the sample to be examined, thus making a dilution of 1 to 100. From it 1 cubic centimeter was removed and added to the second flask containing 9 cubic centimeters, making a second dilution of 1 to 1,000. By a continuance of this method, namely, the removal of 1 cubic centimeter and its addition to the fresh flask containing 9 cubic centimeters of pure water, the dilutions may be run as high as desired. For the routine of this work dilutions of 1 to 1,000; 1 to 10,000; 1 to 100,000, and 1 to 1,000,000, were adopted.

The sowing of the organisms on the nutrient jelly was made by the removal of 1 cubic centimeter from the flask containing the desired dilution and its transference to a sterile petri plate, into which was immediately poured the melted medium and the organisms evenly distributed by shaking with a rotary motion. Duplicate plates were made in all cases and 2 per cent lactose agar was selected as the nutrient medium affording most satisfactory results. All the plates for this investigation were grown at a temperature of 30° C. for a period of three days, after which the colonies when numerous were counted by means of a Stewart counting chamber or when but few by the naked eye alone.

The presence of gas-producing organisms was determined in this investigation by adding 1 cubic centimeter of the 1 to 100 dilution to sterilized 2 per cent dextrose fermentation tubes and incubating at 30° C. for three days. When gas formation took place the quantity was estimated by the ruled scale method, as described by Frost in his Laboratory Manual.

An endeavor was made to determine systematically the presence and approximate number of streptococci in each sample of ice cream, cream and milk, which has been examined recently by this Department. For this purpose 15 cubic centimeters were centrifugalized with an electric centrifuge for a period of fifteen to twenty minutes, and from the sediment were made several smears which were stained with methylene blue. Such a procedure yielded results with milk and cream alone, but when in the form of ice cream, especially those with fruit or chocolate flavors, the débris seemed to interfere to such an extent that satisfactory results were not always obtained. With the vanilla flavors the results were better, but even in such cases they were exceedingly rough. Hence there are a number of blanks in the tables and summaries of this work dealing with the presence in ice cream of streptococci, and the determination of the number of leucocytes per cubic centimeter in ice cream was made in but a few cases.

Between October 13, 1906, and July 29, 1907, 263 samples of ice cream, collected in the City of Washington, were investigated as above outlined. That the bacterial flora in the majority of these ice creams was numerically enormous may be gleaned from the following summary:

Samples showing—	
Less than 10,000 organisms per cubic centimeter	0
From 10,000 to 50,000 organisms per cubic centimeter	0
From 50,000 to 100,000 organisms per cubic centimeter	0
From 100,000 to 250,000 organisms per cubic centimeter	2
From 250,000 to 500,000 organisms per cubic centimeter	3
From 500,000 to 1,000,000 organisms per cubic centimeter	14
From 1,000,000 to 2,000,000 organisms per cubic centimeter	23
From 2,000,000 to 5,000,000 organisms per cubic centimeter	34
From 5,000,000 to 10,000,000 organisms per cubic centimeter	50
From 10,000,000 to 25,000,000 organisms per cubic centimeter	64
From 25,000,000 to 50,000,000 organisms per cubic centimeter	42
From 50,000,000 to 100,000,000 organisms per cubic centimeter	15
Above 100,000,000 organisms per cubic centimeter	16

A study of the individual results from which the above summary was made shows that the average number of organisms per cubic centimeter is 26,612,371. The maximum count obtained was 365,000,000, the minimum 137,500 per cubic centimeter. Of the total number of samples, 71.1 per cent showed the presence of gas-producing organisms when 2 per cent dextrose fermentation tubes were inoculated with 0.01 cubic centimeter of the sample.

Reports on the presence or absence of streptococci have been made on 115 of the above samples; 38.3 per cent of this number showed the presence of the organism, and 61.7 per cent of the samples examined failed to show it when tested by the method above described.

During the course of this investigation 53 manufactories of ice cream in Washington, large and small, have been visited in order to determine the sanitary conditions prevailing where this food product is manufactured. In 62.2 per cent of these places the ice cream is made in the basement or cellar. In nearly all cases they are improperly constructed to meet the demand of sanitary conditions. The ceilings are low and generally show a gross collection of filth and cobwebs on the rough joints overhead. Occasionally a cellar is finished with a metal ceiling or plaster, but even when such improvements are noticed the absence of natural proper light or ventilation generally makes the cellar basement in Washington an unfit place for the manufacture or preparation of ice cream. Many of the buildings are of old-time construction and were not originally designed for the present-day purposes. With such construction as they show it is practically impossible to keep the average basement or cellar in a proper and fit condition for the handling of milk, cream, and milk products, no matter how honest and thorough may be the attempts of the tenants to do so.

In many cases the tenants have much to contend with and report that their landlords are wholly unwilling to make alterations or necessary improvements, and if such are made it must be done entirely at the expense of the tenant. Sometimes, however, the fault does not lie exclusively with the landlord. Very frequently the basement in these establishments is used not only for the manufacture of ice cream and frozen dainties but also as a storage room for all the old waste which may have accumulated for years past—old broken furniture, scraps of metal, cast-off clothing, broken boxes, barrels, moth-eaten rugs, matting—in fact one may find just such worthless stuff as generally collects about the dwelling house in the course of time. Such articles must of course pollute, and most dangerously, any food products which are brought into their proximity, and the nature of the bacterial flora found in the foodstuffs manufactured in these insanitary surroundings fully bear out the truth of the above statement.

While the premises are themselves of insanitary construction an immense benefit would accrue to the consumers of ice creams, charlotte russes, cream puffs, custards, etc., if a general house cleaning on the part of the tenants were demanded and enforced.

An analysis of the individual findings in the 53 places visited and the classification, so far as possible, on the basis of " clean, dirty, fair, and filthy " shows the following results:

Clean.	Fair.	Dirty.	Filthy.
3	16	19	9
[a]5.6	[a]30.1	[a]35.8	[a]16.9

[a] Per cent.

While undoubtedly the insanitary conditions prevailing in and about the ice cream manufactories of Washington must influence the wholesomeness of the product from the bacteriological point of view, it is not entirely responsible for the great number of organisms which are ordinarily found in such foods. As previously stated, the cream and milk supply of the city has been investigated by the Bureau of Chemistry, and although the detailed results will not be reported here, it is advisable to consider briefly the findings of the bacteriological examination of 130 samples of cream collected in the city of Washington from February 1 to July 27, 1907.

Samples showing—	
Less than 10,000 organisms per cubic centimeter	0
From 10,000 to 50,000 organisms per cubic centimeter	3
From 50,000 to 100,000 organisms per cubic centimeter	6
From 100,000 to 250,000 organisms per cubic centimeter	20
From 250,000 to 500,000 organisms per cubic centimeter	19
From 500,000 to 1,000,000 organisms per cubic centimeter	15

Samples showing —

From 1,000,000 to 2,000,000 organisms per cubic centimeter	13
From 2,000,000 to 5,000,000 organisms per cubic centimeter	11
From 5,000,000 to 10,000,000 organisms per cubic centimeter	10
From 10,000,000 to 25,000,000 organisms per cubic centimeter	14
From 25,000,000 to 50,000,000 organisms per cubic centimeter	10
From 50,000,000 to 100,000,000 organisms per cubic centimeter	7
100,000,000 or above organisms per cubic centimeter	2

The preceding summary indicates but too plainly the source of the majority of the organisms in ice cream. Not a single sample showed less than 10,000 organisms per cubic centimeter and only 3 were less than 50,000, while 14, or 10.8 per cent, were between 10,000,000 and 25,000,000. The average number of organisms for all the samples examined was 12,130,080 per cubic centimeter. The maximum count was 309,000,000 and the minimum was 12,000 per cubic centimeter. An examination of these creams for the presence of fermenting organisms showed that when 2 per cent dextrose fermentation tubes were inoculated with 0.01 cubic centimeter 51.53 per cent of the samples developed gas.

Between January 12 and July 2, 1907, a bacteriological examination was made of 381 samples of milk collected in the city of Washington. The quantitative bacteriological findings are appended:

Samples showing—

Less than 10,000 organisms per cubic centimeter	12
From 10,000 to 50,000 organisms per cubic centimeter	59
From 50,000 to 100,000 organisms per cubic centimeter	65
From 100,000 to 250,000 organisms per cubic centimeter	70
From 250,000 to 500,000 organisms per cubic centimeter	40
From 500,000 to 1,000,000 organisms per cubic centimeter	23
From 1,000,000 to 2,000,000 organisms per cubic centimeter	25
From 2,000,000 to 5,000,000 organisms per cubic centimeter	38
From 5,000,000 to 25,000,000 organisms per cubic centimeter	26
From 10,000,000 to 50,000,000 organisms per cubic centimeter	13
From 25,000,000 to 100,000,000 organisms per cubic centimeter	4
From 50,000,000 to 100,000,000 organisms per cubic centimeter	2
Above 100,000,000 organisms per cubic centimeter	2

It was found that the average number of organisms per cubic centimeter was 3,415,533, with a maximum count of 283,000,000 per cubic centimeter and a minimum of 1,000. It is of interest to note, however, that only 12 of the 381 samples showed a bacterial count of less than 10,000. Thirty-seven per cent of the samples showed the presence of gas-producing organisms when tested according to the method previously given.

The foregoing investigations would seem to clearly demonstrate that so far as the ice cream supply of the city of Washington is concerned there is, bacterially, a wide field for its betterment, beginning with the cream and milk which enter into its composition and progressing steadily through every step of its manufacture to the final cleansing of the hands and garments of the employees who dispense this easily polluted foodstuff.

Unfortunately for the good of the country at large, and judging from a cursory knowledge of ice cream manufactories in general and the reported findings of milk and cream supplies throughout the country, the conditions prevailing in Washington can not be accepted as unique.

A study of the commercial ice cream of Philadelphia was made in the Bacteriological Laboratory of the city during 1905–6. (Bacteriological Study of Commercial Ice Cream, Pennington and Walter, New York Medical Journal, Vol. LXXXVI, No. 22, page 1013.) The examination in Philadelphia covered the number of organisms present, an approximate count of the leucocytes, the presence of streptococci morphologically and the determination of their vegetative ability, the sanitary condition of the premises on which the ice cream was manufactured, the sanitary condition of the shop or dealer's warehouse from which the cream and milk were obtained, and the bacteriological examination both numerically and for the presence of living streptococci in the cream and milk which entered into the sample of ice cream studied.

In so far as the cleanliness of the premises and the product is concerned the above authors make the following statements:

> Sixty different ice cream makers were visited and their premises inspected. What constitutes a standard of cleanliness in the production of such foodstuff as ice cream depends very largely upon the inspector's ideas on the subject. The very nature of the process—the mixture of ice and salt, wooden tubs for freezing, fruit flavoring, etc.—makes it a difficult matter to preserve immaculate surroundings even when interiors of utensils and constituents of the ice creams are strictly clean. The final division of these 60 different makers' establishments was made on the basis of four classes: (1) Clean; (2) fair; (3) dirty; (4) filthy. In rating them the building, drainage, opportunities for ventilation, conditions of walls, ceilings, windows, adjoining rooms or buildings, as well as the condition of the utensils, methods of cleaning, attempts at sterilization, etc., were taken into account. The results are as indicated.

Division of 60 different establishments.

Condition	Number of establishments.	Percentage having streptococci in ice cream.	Average count of organisms per cubic centimeter.
Clean	20	90	12, 160, 863
Fair	26	77	15, 857, 800
Dirty	6	66	22, 491, 833
Filthy	8	75	29, 225, 714

The maximum number of organisms found was 151,200,000 per cubic centimeter and the minimum was 50,000 per cubic centimeter.

While the cleanliness of the manufactory does not, according to this investigation, bear any constant relation to the presence of streptococci it does affect the cleanliness of the finished product as indicated by the total bacterial content, a gradual rise being observed from the "clean" shops to the "filthy" ones. The latter were sometimes almost beyond description. For instance, sample 42 was made in a shed adjoining both a dwelling and a stable for 8 or 10 horses. The workmen went from horses and stable cleaning to the ice cream shed without restraint, handling the utensils in the latter as necessity demanded, regardless of soiled clothes or hands. Ice cream cans and milk cans stood in a passageway common to both shop and ice cream manufactory, a part of which was bordered on each side by stalls for horses. The stench of this place finally caused complaint from the neighborhood and it was dealt with on the ground of a nuisance. On the other hand a large ice cream manufacturer had endeavored to preserve the strictest cleanliness possible. Employees engaged in ice cream making did no other work and each man had only certain duties or portions of the process assigned to him. He changed his clothing and took a bath when beginning the day's work and clean lockers and plentiful showers were provided to enable the fulfillment of this regulation. The utensils were cleaned with soda and finally placed on a steam table for sterilization. Such precautions resulted in the counts given in samples 27 and 48 and 49, namely, 6,535,000, 33,120,000, and 20,550,000.

Through the courtesy and interest of the head of this ice cream firm a bacteriological study of each step in the process was made possible. The cream in the supply tank was first sampled, a portion was then drawn off by the employee, mixed with the necessary sugar (cane) for sweetening, and a sample of this taken for examination. After adding the vanilla and transferring to the freezing cans it was again sampled, and then the frozen product was also examined. In the freezing the bulk a little more than doubled. Although frozen the ice cream was soft enough to measure in a wide-mouthed 10 cubic centimeter pipette, and it was plated, after appropriate dilution, at once. The results of the frozen cream, to be comparable with those of the preceding samples, should, therefore, be about doubled. The plates were of agar and were grown at 20° C.

Organisms in ice cream at each step in the process of making.

Articles.	On agar at 20° C., organisms per cubic centimeter.	Streptococci.
Cream from tank	2,840,000	Present, about 25 per cent of all organisms and in an active condition.
Cream and sugar	7,000,000	
Cream, sugar and vanilla in freezer	5,750,000	
Frozen cream	[a] 2,250,000	

[a] Multiplied by 2 equals 4,500,000

It is of interest to note in the examination of the above sample of ice cream that a careful pasteurization had been performed by the ice-cream maker immediately upon the receipt of the cream.

The presence of streptococci in the ice cream on sale in the city of Philadelphia has been made the subject of special study in the article to which reference has been made. The summary of the results states that—

> In 55 out of the 68 samples, or 80 per cent, streptococci were found.
>
> In 45 examinations, or 66 per cent, not only the finished product, but the milk or cream used in its manufacture were investigated. In 35 of the 45 cases, or 77 per cent, streptococci were found in the milk or cream and in the ice cream as well. From 23, or 33 per cent of all examined, the streptococci were isolated in pure culture. They grew fairly easily. In only 3 samples were these organisms found in the cream alone, and where both cream and ice cream were examined only twice in the ice cream alone. The question of the original source of streptococci in ice cream is of importance from a sanitary standpoint. The conditions under which the mixtures are made and frozen, the cleansing of the utensils, etc., are such that very often almost any kind of bacterial infection may gain access to it.
>
> The usual source of streptococci in milk or cream, however, is the cow, and, judging from the results set forth here, it is the cream or milk entering into the ice cream which is the carrier of the germs. The cleanliness of the surroundings under which the ice cream is made does not seem to greatly affect the presence of streptococci.

Since ice cream is a food which is so largely used by children and invalids whose digestive tracts are more readily open to bacterial infection than are those of the adult or the person in perfect health, the widespread presence of an organism to which so much responsibility for ill-doing is attached as appertains to the streptococcus, should be looked upon with suspicion and every care possible taken to exclude it from such food products—at least until it has been proven innocuous.

There seems to be a certain class of adults who have a predisposition against ice cream and who can not ingest it without a feeling of discomfort and in not a few cases symptoms of severe toxic poisoning result, manifesting the usual course of nausea, vomiting, diarrhea and pains in the abdomen, with cramps and muscular pains, often followed for a short time by general weakness, malaise, loss of appetite, and headache. Where samples of ice cream associated with such disturbances have been examined bacteriologically they have often shown the presence of overwhelming numbers of streptococci, constituting practically a pure culture, or associated with organisms such as *B. coli* or other bacteria known to be found under insanitary conditions.

Where in the routine examination of a city's milk supply the absence or presence of streptococci is made the subject of investigation, it has been found that approximately 40 per cent of the milk offered commercially contains these organisms, and in the cases of certain individual cities the results are much higher. According to the investigations, already quoted by Pennington & Walter, 80 per cent of commercial ice cream contains these organisms. In an endeavor to determine the reason for this high frequency, they conducted a study on the relative rate of growth of streptococci isolated from milk, in milk and cream, and find that there is a much more rapid proliferation of the organism in cream than in milk. The difference in the relative rate of growth is more striking, also, at the temperature of the refrigerator (about 12° C.) than at higher temperatures, which may account, at least to some extent, for the frequency with which this organism occurs in ice cream and also for its overwhelming proportion there.

It was noticed also that the thickening of cream, inoculated with pure cultures of streptococci and kept cool, was very marked. Its whipping quality greatly increased and the separation of a curd was extremely slow, all of which qualities are sought after by the ice-cream maker.

CHANGES IN ICE CREAM DURING STORAGE.

An important point to be considered in the study of ice cream is the change which takes place during the storage thereof. It is quite customary at the present time to make a kind of ice cream which is intended to be kept a long while and shipped to great distances. It is generally supposed that very low temperatures entirely inhibit bacterial growth. That this is not always the case is shown by the results of the investigations which are appended. In order that some definite knowledge might be obtained of what actually takes place respecting the bacterial flora during cold storage two sets of investigations were instituted—one in Washington under the supervision of Dr. George W. Stiles, and one in Philadelphia under the direction of Dr. M. E. Pennington. Doctor Stiles's report is as follows:

The technique used in the study of ice-cream samples kept in a frozen condition for about thirty days corresponded very closely to that used in the quantitative examination of the ice-cream samples heretofore described. The sampling, however, was of necessity somewhat modified.

From each of four representative dealers twelve 5-cent samples were purchased, each sample being kept separate in a 5-cent paper carton, as used by such dealers. One dealer, however, not having the small cartons at hand wrapped the samples each in tissue paper and placed all of them within a new pasteboard box. The samples were kept in a cold-storage warehouse where the temperature varied from 0° to 10° above 0 F. The graphic chart shows the variations in bacterial content of these four groups of samples.

In addition to making counts of the number of organisms present, each sample was tested for gas-producing organisms, and from each a bacillus was isolated which belonged to the *B. coli* group.

The initial count of sample No. 1 was 16,000,000; of No. 2, 85,000,000; of No. 3, 135,000,000, and of No. 4, 53,000,000. The variation from these numbers during the keeping of the sample will be noted in the table which follows, as well as the decrease of gas production in some and no noticeable difference in others, especially No. 1, which showed gas-producing bacteria during the entire

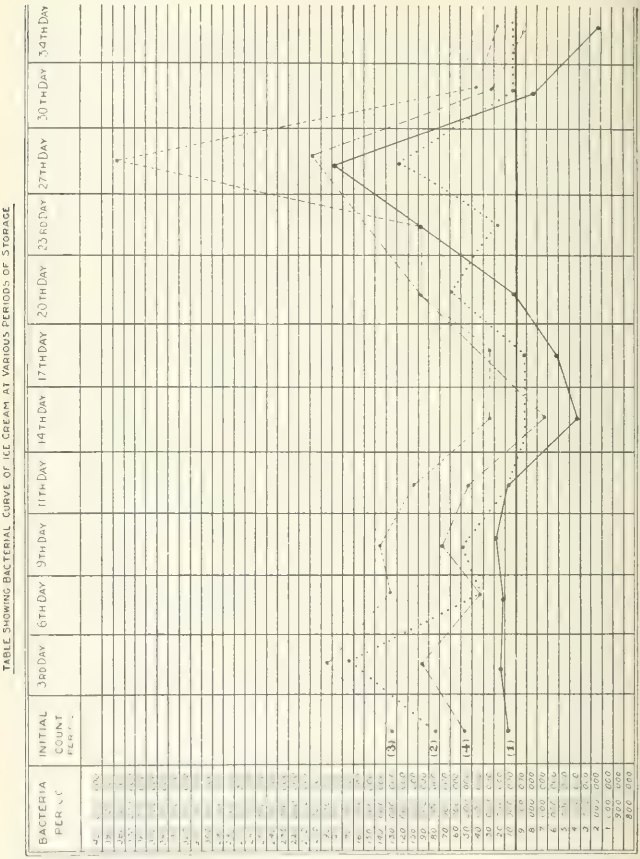

FIG. 1.—Variations in bacterial content during cold storage of four samples of commercial ice creams (Washington).

period. With a decrease in the gas producing bacteria after the first or second week there was also noted a marked decrease in the number of organisms, though in many cases these again increase.

Table showing the number of organisms and percentage of gas production in cold-storage ice cream.

Group No.	Serial number of sample.	Days in storage.	Bacteria per cubic centimeter.	Percentage of gas.	Percentage of gas, duplicate.
1	401	0	16,000,000	25	25
	429	3	25,000,000	45	35
	433	6	21,000,000	30	30
	440	9	27,000,000	20	30
	484	11	18,000,000	25	25
	489	14	3,000,000	25	20
	542	17	6,000,000	30	35
	538	20	13,000,000	30	25
	546	23	20,000,000	20	25
	555	27	185,000,000	20	20
	564	30	8,000,000	15	10
	568	34	3,000,000	20	20
2	404	0	85,000,000	25	25
	430	3	175,000,000	5	5
	434	6	38,000,000	0	0
	441	9	18,000,000	30	0
	485	11	18,000,000	0	0
	490	14	9,000,000	0	0
	543	17	9,000,000	0	0
	539	20	70,000,000	0	0
	547	23	19,000,000	0	0
	556	27	125,000,000	2	0
	565	30	10,000,000	0	0
	569	34	11,000,000	0	0
3	403	0	135,000,000	25	25
	431	3	195,000,000	30	20
	435	6	138,000,000	20	30
	442	9	145,000,000	30	40
	486	11	115,000,000	40	25
	491	14	39,000,000	2	8
	544	17	35,000,000	9	10
	540	20	93,000,000	1	0
	548	23	97,000,000	5	5
	557	27	385,000,000	18	0
	566	30	50,000,000	18	0
	570	34	28,000,000	0	0
4	402	0	63,000,000	25	10
	432	3	95,000,000	18	20
	436	6	47,000,000	25	15
	443	9	75,000,000	10	20
	487	11	54,000,000	15	1
	492	14	7,000,000	1	0
	545	17	27,000,000	9	20
	541	20	95,000,000	0	0
	549	23	19,000,000	1	0
	558	27	205,000,000	5	0
	567	30	30,000,000	0	0
	571	34	9,000,000	12	0

The experiment conducted under the direction of Doctor Pennington follows:

While the temperature of 0° C. is ordinarily accepted as that at which bacterial life is either quiescent or annihilated, the making of ice cream in a freezing mixture of ice and salt reduces the substances to a temperature of from —10° C. to —20° C. To test the action of this temperature on the very rich bacterial flora ordinarily occuring in commercial ice creams, samples were obtained from various sources and maintained for several days at a temperature varying at from —10° C. to —20° C. It has been found by inquiry and observation that ice cream may be kept by a manufactory or more likely by the retail dealer for a week or ten days. It is of course kept for greater lengths of time when provisioning ocean liners or stored for some particular purpose, but this is rather the exception. It was deemed advisable, therefore, to limit this preliminary investigation to the period which is ordinarily that of commerce.

The samples of ice cream tested were purchased in open market or sent directly from the manufacturer, who had no knowledge of the purpose for which they were intended. When coming from the manufacturer they were packed in the usual tin ice-cream storage can, set in an ice and salt mixture. When purchased from restaurants, confectioners, etc., the sample was obtained in a sterile wide-mouthed glass jar, tightly capped, and was immediately packed down in ice and salt. All the samples to facilitate keeping were placed in a room in the cold-storage warehouse, where the temperature was slightly below freezing. Here they were inspected daily, ice and salt added as required, and samples for study removed with sterile glass spatulas.

For the determination of the total number of organisms, approximately 1 cubic centimeter of the cream, which was melted as promptly as possible after reaching the laboratory, was weighed in a tared, sterile weighing bottle, made up to 10 cubic centimeters with sterile water, and from this mixture were prepared appropriate dilutions for the counting of organisms.

The plating was done on litmus lactose agar, half the plates of each sample being allowed to develop colonies at a temperature of 37° C., and the other half placed in the refrigerator, running from 18° C. to 20° C. It has been found necessary to use both these temperatures if an accurate idea is to be obtained of the changes undergone by organisms when submitted to continuous low temperatures, since there is apparently a dying off of certain groups of organisms in the early stages of storage, and the gradual increase of other organisms, which seem ultimately to thrive under what are usually conditions fatal to growth.

The results obtained in the study of 8 samples of ice cream are tabulated as Series I, II, and III, of Table I. Series I extended over sixty-six hours; Seres II covered a total of one hundred and ninety-two hours, and Series III a total of two hundred and sixteen hours.

As with all experiments where the bacterial flora is of as varied a character as that found in commercial cream, there is observed in this work a considerable variation in the behavior of different samples. Generally the tendency is to show a decrease for at least several days in a number of organisms developing at body heat, though occasionally these organisms persist and thrive at low temperatures. The organisms developing at the temperature of the refrigerator show usually a period of decrease which may last several days, to be followed later by a very pronounced rise. Sometimes the killing off of the organisms is very slight, their numbers remaining almost stationary or making a continuous upward curve.

The experiments cited are too few to permit of formulating definite conclusions and it must not be forgotten that, purchased as they were—in open market—their histories unknown, there may have occurred a considerable part of the bacteriological cycle before the specimens were investigated. The results given simply show what may happen to commercial ice cream if kept for from three to ten days.

Two experiments have been made to test the action of freezing and thawing. For this work cream was obtained from a milk dealer, sweetened and flavored with vanilla in the laboratory, and frozen in a small hand freezer, which had been cleansed simply with hot water. After freezing the product was packed down in an ice-salt mixture and allowed to stand until the cream had thoroughly melted, though the temperature was still considerably below that of the surrounding atmosphere. It was then refrozen and again allowed to stand for some hours. At each step the bacterial count was made and recorded, as seen in Table II. Experiments of this character are of interest, not only for the tracing of decrease in numbers, but also as a possible source of information regarding the many cases of ice-cream poisoning blamed upon stale ice cream, and particularly that which had been melted and refrozen.

TABLE I.—*Growth of bacteria in ice cream at the temperature of ice and salt (−21° C.=−5.8° F.).*

SERIES I.

Source of ice cream.	Total number of organisms in—			
	1 gram of fresh ice cream.	1 gram after 18 hours.	1 gram after 42 hours.	1 gram after 66 hours.
No. 1—Manufacturer, sample sent directly from factory	a811,249 b2,523,886	a1,010,509 b1,010,509	a3,349,733 b628,074	a4,405,286 b1,664,218
No. 2—Manufacturer, sample sent directly from factory	a4,142,068 b9,521,995	a2,552,676 b1,495,066	a2,603,421 b464,000	a4,550,050 b3,993,933
No. 3—Manufacturer, sample sent directly from factory	a3,375,527 b9,493,670	a4,173,622 b936,065	a1,055,966 b422,386	a4,264,870 b16,835,016

SERIES II.

Source of ice cream.	Total number of organisms in—				
	1 gram of fresh ice cream.	1 gram after 24 hours.	1 gram after 48 hours.	1 gram after 96 hours.	1 gram after 192 hours.
No. 4—Low grade confectioner	a564,381 b1,097,408	a164,000 b201,786	a140,814 b1,804,180	a149,812 b2,536,115	a61,312 b1,021,867
No. 5—Wholesale milk company and dairy lunch	a1,006,904 b5,418,105	a1,489,361 b2,765,957	a1,171,613 b2,454,809	a816,405 b3,936,242	……… …..

SERIES III.

Source of ice cream.	Total number of organisms in—					
	1 gram of fresh ice cream.	1 gram after 24 hours.	1 gram after 48 hours.	1 gram after 72 hours.	1 gram after 120 hours.	1 gram after 216 hours.
No. 6.—Cheap restaurant which buys from manufacturer of sample No. 1.	a2,668,777 b6,219,608	a298,804 b1,090,637	a709,442 b855,503	a690,000 b1,189,296	a292,839 b2,352,163	a1[illegible],351 b946,611
No. 7.—Small bakery, premises clean	a12,460,196 b29,558,355	a3,231,017 b2,670,974	a4,019,523 b8,010,335	a12,735,849 b2,452,830	a8,052,455 b2,300,701	a644,329 b5,257,731
No. 8.—Market house lunch counter, ice cream made by proprietor.	a510,673 b714,942	a1,256,645 b2,319,961	a1,424,936 b1,323,1[illegible]	a776,196 b217,33[illegible]	a476,558 b1,449,725	a49,499 b1,005,615

a 37° C.   b 18° to 20° C.

TABLE II.—*Bacterial growth in ice cream thawed and refrozen.*

Source of ice cream.	Total number of organisms in—				
	1 gram of cream.	1 gram of ice cream.	1 gram of melted ice cream 14 hours after freezing.	1 gram of refrozen cream.	1 gram of ice cream 7 hours after refreezing.
Experiment 1—Home-made, vanilla	[a]1,142,640 [b]1,158,886	[a]274,254 [b]351,219	[a]57,090 [b]331,125	[a]32,829 [b]306,412	[a]28,774 [b]32,696

Source of ice cream.	Total number of organisms in—				
	1 gram of cream.	1 gram of ice cream.	1 gram of melted ice cream 23 hours after freezing.	1 gram of refrozen cream.	1 gram of ice cream 5 hours after refreezing.
Experiment 2—Home made, vanilla	[a]2,926,421 [b]7,525,125	[a]1,144,016 [b]3,874,896	[a]897,867 [b]2,244,668	[a]614,463 [b]732,629	[a]1,541,501 ....

[a] 37° C.

[b] 18° to 20° C.

All of the samples which have been studied for cold storage history were examined also for the presence of streptococci. The results are indicated in the following list:

Number.	Streptococci.	Number.	Streptococci.
1	Not found.	5	Present—short chains—few.
2	Present—short chains—numerous.	6	Present—long chains—numerous.
3	Present—long chains—very numerous.	7	Not found.
4	Present—short chains—few.	8	Not found.

Eighty-two and five-tenths per cent showed the presence of the organism.

The method for the detection of streptococci in ice cream was as follows: The melted sample was centrifuged for half an hour in a Stewart lactocrite driven by a small motor of such power that the speed was approximately 3,000 revolutions per minute. This apparatus, which consists of a flat aluminum pan holding 20 tubes of 1 cubic centimeter capacity and stoppered at the outer end with a specially constructed rubber plug, causes the sediment not only to be thrown to the end of the tube but drives it against the rubber plug with such force it is almost quantitatively adherent to the plug. Accordingly, if one carefully removes the rubber stopper and by rubbing on a glass slide and over an area of known surface attaches the sediment, one can obtain, on staining and examining the film microscopically, an approximation of the number of organisms and leucocytes in 1 cubic centimeter of the liquid.

Because of the débris in ice cream, which ordinarily renders the usual method of centrifuging milk and cream samples quite impracticable, the above method was resorted to and, so far as the detection of the presence of streptococci was concerned, it was found eminently satisfactory.

THE SIGNIFICANCE OF A PURE ICE CREAM SUPPLY IN RELATION TO PUBLIC HEALTH.

A study of the literature dealing with diseases traced to the eating of ice cream shows that not only are isolated cases more or less severe, even sometimes resulting in death, fairly numerous, but widespread epidemics have been caused by the toxicity of the substance. Such diseases are, of course, of gastro-intestinal origin. Among these epidemics is one of typhoid fever described by Dr. George Turner, occurring at Depford in 1891, which was apparently caused by ice cream.

Another epidemic of this disease occurred in Liverpool in 1897 to which 27 cases were traced.

In 1902, in the city of London, 18 cases of typhoid fever were traced by the health officer of Finsbury (see report of health of Finsbury, 1902, page 67) to ice cream as the source of infection.

More commonly, however, the illness caused by ice cream has the symptoms of colic, headache, diarrhea, and depression rather than a specific typhoid infection. "Such an outbreak occurred in Birmingham during the summer of 1905 (Thresh & Porter, Preservatives in Foods and Food Examination, page 280) and was investigated by Dr. Robertson, the city medical officer of health. Out of 250 consumers served 52 cases of illness occurred, 4 only of the patients being over 14 years of age. The interval which elapsed between the eating of the ice cream and the onset of the illness varied from half an hour to eight and a half hours. All the persons suffered from diarrhea and collapse. No irritant poison was discoverable by chemical analysis. Professor Leith examined the ice cream bacteriologically and found therein a bacillus of the colon group capable of causing the death of guinea pigs. From an examination of the premises in which the ice cream was manufactured it appeared probable that it had become contaminated while standing in the cooling shed after boiling and before freezing. Opposite this shed there were 3 waterclosets in an extremely filthy condition, and possibly organisms of excremental origin had fallen upon one of the buckets of the cream while it was in a warm condition. These would rapidly multiply and may have produced toxins or ptomaines. Neither the bacilli nor their poisonous products would be affected by the subsequent freezing."

In the discussion of ice cream in "Bacteriology and Public Health," by George Newman, he states that a "small outbreak occurred in the city of London, affecting 16 telegraph boys. The symptoms were colic and diffuse abdominal pains, headache, vomiting, diarrhea, and nervous depression. Dr. Collingridge's inquiry resulted in the following conclusions:

(1) That in a number of cases of illness occurring among young persons of a susceptible age the symptoms were strictly identical and were characteristic of poisoning by ingestion of toxic material.

(2) That the cases reported followed the ingestion of ice creams.

(3) That ice creams subsequently obtained at shops frequented by the patients contained bacilli of a virulent character.

(4) That the symptoms observed were those generally following the ingestion of material containing such bacilli.

(5) That where pathogenic bacilli were found, the ices had been manufactured under insanitary conditions. The majority of the manufacturers are aliens, and although the premises may be kept in a fairly sanitary condition, their personal habits unfortunately leave much to be desired where the preparation of food is concerned."

Dr. Klein examined 24 samples of ice cream from the same locality and found 13, or 54 per cent, to be poisonous to guinea pigs.

In July of 1904 the medical officer of health of Battersea (Report of 1904, Public Health Committee of the London County Council) reported an outbreak of illness among the people who had eaten ice cream purchased at a particular shop. As usual in such toxemias the symptoms included abdominal pain, diarrhea, and collapse. The ice cream causing these poisonings had all been eaten and therefore could not be examined, but an inspection of the premises showed very filthy conditions and in all probability the contamination of the cream was due to a dust bin in the immediate proximity of the shelf on which the ice-cream vessels were stored.

Owing to outbreaks of this nature the London County Council (general powers act, 1902, secs. 42–45) has given powers for controlling this trade:

(*a*) Ice cream must be made and stored in sanitary premises.
(*b*) It must not be made or stored in living rooms.
(*c*) Strict precautions must be taken as to protection from contamination.
(*d*) Cases of infectious disease must be reported.
(*e*) The name and address of the maker must appear on street barrows.

These regulations are new for London, though they have practically been in existence in Glasgow since 1905, and in Liverpool since 1898.

That such powers are enforced by the officials having the public health of London in charge is demonstrated by the report, for example, of the sanitary conditions relating to the city of Westminster for the year 1903. (Francis J. Allan, medical officer of health for the city of Westminster.)

> Premises where ice creams are manufactured or sold were frequently inspected during the year; there were 108 premises other than hotels and restaurants where ice cream is manufactured and sold. Proceedings were taken against Pietro Necchi, 36, Berwick street, under the London County Council general powers act, 1902, for manufacturing ice cream in a room used as a sleeping room, and he was fined £2 and £2 2s. costs.
>
> Every itinerant vendor of ice cream, etc., is required to exhibit the name and address of the manufacturer on his barrow. One man was cautioned under this section. Lists of such vendors were prepared in several boroughs, and the medical officers of health gave one another information with regard to the places where the ice cream is made. There were 18 persons selling it in the city during the year, of these several resided in the city (of Westminster), the others came from Finsbury (4), Holborn (3), Chelsea (3), and Lambeth (1). In one case the medical officer of Chelsea informed me that the place in which the mixture was prepared was dilapidated, with water-closet obstructed, and defective paving of scullery. In another (in Holborn) proceedings were taken for making ice cream in a living and sleeping room.

The significance of the streptococci as a disease-producing organism in ice cream has been briefly discussed in the section of this report devoted to bacteriological findings of ice cream in the city of Washington, and to that section the reader is referred. Aside from the invasion of the organism by living pathogenic bacteria, and the characteristic symptoms following such invasion, there must not be forgotten the causation of illness by products of the bacteria themselves—even though they as living cells may have been eliminated either by boiling, freezing, or the use of chemical preservatives. It is commonly supposed that the manipulation through which the mixtures for ice creams are apt to go, namely, pasteurization or scalding of at least a portion of the ingredients would tend to lessen the actual number of organisms present and to kill those which are commonly considered to be pathogenic. So far as the lessening of the number of organisms is concerned, the investigations embodied in this report offer an emphatic denial; and the heat or cold to which the mixture is exposed would be absolutely without effect upon the toxins or ptomaines produced by the organisms even should the latter be killed.

Indeed, it may be very seriously questioned whether preliminary heating of the milk products going into the compounds known as "ice cream" is not actually deleterious and responsible, to some extent, at least, for such cases of poisoning as are included under the popular term of "tyrotoxicon." It has been definitely established that the scalding or commercial pasteurization of milk and cream of the usual commercial quality tends to kill off the organisms producing lactic acid and naturally causing the milk to curd, but leaves behind the organisms more resistent to heating and which are apt to be those forming, as part of their excreted product, alkaline substances, which—as the acid forming organisms are not there to give them combat—increase to such an extent that the reaction of the milk itself becomes distinctly alkaline.

Ptomaines are chemical substances built on the ammonia type and are most commonly produced by bacteria coincidental with an alkaline reaction, or in a medium which has previously been made alkaline in reaction. Any condition therefore which produces in milk or its products circumstances favorable to the production of ptomaines is undesirable. The fact that the great majority of reported cases of ice cream poisoning are to be traced to the use of cheap grades of material would tend to confirm the foregoing supposition, since these cheaper grades of ice creams are commonly made of milk, eggs, gelatin, and such thickeners as require heating in order to produce the desired result.

The use of condensed milk in cheap grades of ice cream is by no means uncommon. Indeed, with the increased activity of the condensed-milk agent and the increased demand—particularly in large cities—for fresh milk, the practice is growing more and more popular, and such condensed milks and those substances known as "evaporated creams," which are only whole milks concentrated, are far too apt to usurp the place of true cream in the manufacture of ice creams.

The contention has been raised by the makers of ice creams that the proposed Federal standard of butter fats is too high to be healthful and that an ice cream containing the amount of cream required by the Federal law can not be digested by many people. They assert, however, that an ice cream containing milk, eggs, and sugar, with such a thickener as cornstarch or gelatin, can be digested by, and is grateful to, those with whom the true ice cream does not agree. It is widely known, however, among those who have had experience in the feeding of invalids, convalescents, and persons having impaired digestive organs, that the unification of 3 of our most concentrated foods—such as milk, eggs, and cane sugar—produces a combination which is difficult of digestion and the feeding of it is often impossible. In such cases the patient can assimilate either of the ingredients separately, or any two in combination, but the third concentrated food when mixed with the other two is more than the organs can metabolize.

Such being the case it would seem doubly desirable from the standpoint of the physician and the hygienist that there should be on the market a standard preparation consisting exclusively of cream, sugar, and flavoring, and of a definite fat content, that he may know what is being fed to his patient.

The correct labeling of the frozen mixtures sold at retail would also enable the person, who by experience has found that pure cream ice cream is or is not suited to his digestive organs, to obtain that which does agree with him.

Not only is the chemical composition and the bacteriological decomposition of ice creams widely discussed in the literature from the standpoint of food value and desirability, but there comes from Italy an article by Baldoni, in the "Riforma Medica" for 1907, in which he attributes much of the digestive disturbance in Rome during the summer time to the contamination of ices by tin and lead, which are scraped off the inside of the freezing can by the mechanical action of the dasher. Baldoni has not only proven the presence of these metals in ices in a dissolved condition, but by careful filtration he has isolated macroscopic particles of both lead and tin.

The container of ices, etc., commercially, is a metal cylinder, in which products having various fruit flavors are stored for considerable lengths of time. In some cases the material melts, warms up very thoroughly, and is again frozen. It is perfectly possible that a mechanical distribution of particles of metal throughout the mass, and the long-continued action of fruit juices on these small particles as well as on the surface of the container, result in the accumulation in the food stuff itself of very appreciable quantities of metallic salts.

DEFINITIONS AND DESCRIPTIONS OF ICES IN TRADE AND OTHER BOOKS.

In an anonymous work entitled, "Ice Cream and Cakes," by an American, published by Chas. Scribner & Sons, in New York in 1901, the materials for making ice cream are described as follows: "Cream, sugar, eggs, flavors in variety, fruits and their juices, ice and snow, salt. Cream is classified by the author as single, double, and butter cream. Single cream is that which is skimmed from milk twelve hours after milking, double cream twenty-four hours after milking, and butter cream thirty-six hours after milking. No mention is made by this author of cream which is separated mechanically and which practice is now more frequently used for ice cream perhaps than any other. The author states that for making ice cream only the double cream of entire purity should be used and as soon after skimming as possible. On page 15 the author says:

> Milk should not be used, either wholly or in part, in place of cream. Its watery portion freezes into coarse crystals that give a snowy, mushy taste to the ice cream, which even the use of eggs does not correct, and causes it to melt much more rapidly than when made of pure cream.
>
> To prevent this and give the appearance of genuine ice cream some makers put in gelatin, to keep it firm, as they say. But its taste betrays it; neither

gelatin, tapioca, cornstarch, arrowroot, or any other makeshift will compensate for the absence of pure cream.

The use of milk should be discountenanced by all who would have and enjoy ice cream of the best quality. In truth, when made of milk and eggs and not cream the product is frozen milk custard.

By the same author the American type of ice cream is called "Philadelphia," and the following statement is made:

> Perhaps in no place in America can the Philadelphia ice creams be found of higher quality than at a first-class confectioner's in the City of Brotherly Love. Certainly nowhere else can the chief material, pure cream, be obtained of greater richness and more delicious flavor.

This is somewhat misleading, since there are doubtless hundreds of places in the United States where just as good cream with just as fine flavor can be found as in Philadelphia.

The American anonymous author above mentioned recommends the use of eggs in that variety of ice cream known as "Neapolitan" which, however, in its own country is not called a cream. He states that the Neapolitan ice creams do not differ from the Philadelphia creams except in the use of eggs in their composition. Types of Neapolitan ices are made according to the formulas given in the book; the most popular one is called vanilla, No. 53—which is made of 2 quarts of cream, 12 eggs, 1½ pounds sugar, and 1½ ounces of a mixture of 1 pound of sugar with 1 ounce of finely powdered Mexican vanilla bean. In regard to the Philadelphia ice creams the author says, on page 60:

> Although some of the best confectioners in the Quaker City make their creams somewhat after the Neapolitan method, in proportions varying from 6 to 1 egg for 1 quart of cream, some using the whole egg, others the yolk only, yet the plain creams without eggs for which that city has long been famous have become so generally known by its name that the title is here retained as their proper and distinctive designation. There is no other name for them.
>
> The question of the relative qualities of the Neapolitan and Philadelphia creams is one of either education, taste, or comfort. Those who are fond of eggs and custards will prefer the former; those who are partial to pure cream, as well as those with whom eggs do not agree, will choose the latter.

A typical formula for the vanilla cream is also given on page 60 as follows:

> Three quarts of cream, 1½ pounds of sugar, and 1½ ounces of the mixture of sugar with finely powdered Mexican vanilla bean, above described.

Following this recipe are given recipes for chocolate, chocolate caramel, coffee, white coffee, caramel, pistachio, almond pistachio, almond, sweet almond, burnt almond, orgeat, filbert, burnt filbert, hazelnut, walnut, chestnut, lemon, orange, pineapple, banana, strawberry, raspberry, peach, apricot, nectarine, plum, cherry, apple, currant, and grape. In none of these are any components admitted except the cream and sugar, save the proper flavoring matters derived exclusively from the substances mentioned. This classification is by far the most rational and satisfactory of any that I have been able to find in other authors. Nearly all the other authors admit indiscriminately to the name of ice cream all the various compounds which have been described.

Among other authorities of this kind I may mention "Mrs. Lincoln's Boston Cook Book," Roberts Bros., edition of 1897. This author gives the formula for Philadelphia ice cream and Neapolitan ice cream exactly in harmony with the author just quoted. The following naive statement is made on page 363:

> If cream can not be obtained beat the whites of the eggs till foamy, and add them just before freezing. No matter how many eggs are used, a little cream, if not more than half a cupful, is a decided improvement to all ice creams. It is better to make sherbet or fruit and water ices than an inferior quality of ice cream with milk. Ice creams are richer and mold better when made with gelatin, but care must be taken to flavor highly to disguise the taste of the gelatin.

In "Mrs. Rorer's New Cook Book," edition of 1903, it is stated, page 600:

> To make good ice cream it is first necessary to have a good quality of cream. Scald half the cream to prevent excessive swelling. Where fruits are used they must be mashed and added after the cream is frozen.

The formula for peach ice cream admits only cream, the fruit, and granulated sugar. The same is true of strawberry ice cream and raspberry ice cream. In chocolate ice cream as much milk is admitted as there is of cream. For vanilla ice cream nothing is admitted except cream, vanilla bean, and sugar. It is seen, therefore, that Mrs. Rorer upon the whole, with one exception, admits nothing but cream, flavor, and sugar into her products.

Mrs. Mary J. Lincoln and Anna Barrows, in a work entitled "The Home Science Cook Book," describe ice creams and other frozen desserts on page 186 and following. The general name of frozen desserts is given to the whole class. The authors say:

> So many names are given to different frozen desserts that a few words of explanation are needed.
>
> ICE CREAM.
>
> This consists mainly or entirely of cream and takes a specific name from the substance used for flavoring.
>
> FROZEN PUDDING.
>
> Ice cream or custard, highly flavored, and containing preserved fruits and nuts becomes frozen pudding.

In "Mary Ronald's Century Cook Book," edition of 1897, the author includes, p. 188, under the term "Frozen Desserts," ice creams, water ices, parfaits, mousses, frozen fruits, punches, and sherbets. Ice creams are classified as follows:

> Philadelphia ice creams are cream sweetened, flavored, and stirred while freezing.
>
> French ice creams are custards of different degrees of richness stirred while freezing."

Then follow the definitions of parfaits, bisques, and mousses, which are described as whipped cream with or without eggs, frozen without stirring. The author adds:

> These creams, in different degrees of richness and with different flavorings, give an infinite variety, and their combinations and forms of molding give all the fancy ices.

Mrs. Ronald does not mention gelatin as a constituent of either straight ice creams or of any of the frozen custards or desserts which she describes.

In "Paul Richard's Pastry Book," page 78, is found the following:

> The best and richest ice creams are made from double cream, with the addition of yolks of eggs, sugar, and flavorings, while some of the cheapest commercial creams are made from milk only, without eggs, and are thickened with gelatin, corn starch, arrowroot, sago, and other preparations. The rich creams which contain eggs and cream frozen in patent freezers are also termed New York creams, and the lighter creams, made from the best cream and without eggs, Philadelphia creams.

On page 82 it is stated that the name Philadelphia ice cream "is generally applied to ice creams made with pure cream and without any eggs, although some makers use about 5 eggs to each gallon of cream, with 2 pounds of sugar."

Under the head of commercial ice creams the author says:

> Where quantity is more required than quality ice creams are made from plain cream, half milk and half cream, and of milk only. Starch, arrowroot, and sago flour in proportion from 3 to 6 ounces to each gallon is boiled into a smooth batter with a part of the milk and the sugar, strained, cooled, and frozen. Gelatin should be soaked and dissolved in warm milk but not boiled, as this would cause the milk to curdle. About 1½ to 2 ounces of gelatin are used for 1 gallon of cream and milk. Another thickener for ice creams is used cold. The preparation is known to the trade as cream-thick; it is something like a dry milk powder. The thickener is mixed with the sugar to be used, the cold milk or cream added gradually. As soon as the sugar is dissolved the cream is ready to be frozen.

Caterers' standard ice cream, best quality, according to Paul Richards, is made after the following recipe:

> 1 gallon double cream, flavor, 1¼ pounds sugar. The cream is made by the cold process and is used by the best caterers as a standard preparation from which are made many of the fancy creams, fruit, and nut creams.

In the "Ice Cream and Candy Makers' Factory Guide," edition of 1907, are found the following notes on page 4:

Scalding milk or cream means to bring it to the steaming point over hot water; never allow the material to boil.

When part milk is used the cream may be whipped before freezing.

If eggs are used cook them with the milk or cream.

Well beaten white of egg added to a frozen sherbet makes it creamy and smooth; added to any of the creams will make it smoother and lighter.

Good ice cream can be made without cream (part 5).

The Philadelphia, or eggless, cream is best if fruits are to be added.

Cream two or three days old is better than cream one day old.

Scalded cream gives greater "body" and when frozen will have a fine grain.

Ices made with too much sugar are hard to freeze and sometimes "ropy;" if too little sugar is used they will be coarse and rough.

Sour fruits should be added to the cream after it is frozen.

Raspberries, lemons, and oranges make better water ices than ice creams.

On page 5, the author quotes the national standards for ice cream and adds the following comment:

It is generally thought that the standard has been set too high, but it is the law, and is in the right direction, as it protects the public against misrepresentations, and against harmful ingredients; besides it does not prohibit the shipment of creams that differ from the standard, but it simply requires the shipper to designate the actual quality by a label.

On page 8, in describing the ice creams known as "Philadelphia," the author says:

Includes all the various creams made of pure cream, without eggs.

Part 7 of this work is devoted to "commercial" ice cream. The first formula given is that of "fortuna" cream, of which it is stated that this formula made a fortune for its originator. It is as follows:

4 gallons 20 per cent cream, 1 gallon condensed milk, 1 gallon fresh milk, 4 ounces gelatin, 7 pounds granulated sugar, 3 ounces vanilla extract.

Chicago "picnic" ice cream is made as follows:

14 quarts condensed milk, 10 quarts fresh milk, 8 pounds granulated sugar, 8 ounces gelatin, 4 ounces vanilla.

The "economy" formula for ice cream is as follows:

9 gallons fresh milk, 10 pounds granulated sugar, 10 ounces gelatin, 4 ounces cornstarch, 4 ounces vanilla extract.

The author says, after describing how the materials are mixed together:

It is now ready to freeze and when frozen will be smooth and fine grained and appear as if made from cream. It will never be blue and coarse, cheap looking, and cheap tasting, like milk mixtures generally.

The "Chicago" formula for ice cream is as follows:

> 4½ gallons cream, 1 gallon condensed milk, 7 pounds granulated sugar, 6 ounces gelatin, 4 ounces vanilla extract.

After describing the method of mixing and freezing the author says:

> Six gallons of this mixture will make 10 gallons of high-grade ice cream, rich and smooth. The cream should be several days old.

Trade journals devoted to confectionery and ice-cream making have had much to say during the past two or three years respecting ice cream and the method of its manufacture.

In the Confectioners' Journal of May, 1907, page 94, the editor says:

> Now for the ice cream and soda water. Use only the very best materials, and don't make the great mistake of thinking that this and that will do, but resolve that only the very best is just "good enough" and a good business with a fair profit will be the reward. As cream is the most important ingredient in the manufacture of ice cream, we wish to say a few words about the same before we go into details of the manufacture of the ice cream. Cream is classified as follows: Single, double, and butter cream. Single cream is that which is skimmed from milk twelve hours after milking, a "double" cream is allowed to stand twenty-four hours before it is skimmed, while butter cream—which does not come into consideration in this article—stands thirty-six hours before skimming.

Gelatin is not mentioned by the editor as a component of ice cream; he says, however, in speaking of water ices:

> In order to smooth water ice the addition of raw egg white is best, although glucose and gelatin are often used instead.

In the same journal of June, 1907, on page 90, various formulas are given for making different kinds of ice cream and ices. The recipes given for ice cream contain no ingredients except cream and sugar and the flavor. The recipes given are for grape, banana, bisque, pistachio, peach, apricot, filbert, roasted filbert, walnut, pineapple, cherry, and cocoanut ice creams. The formula for Neapolitan ice cream, however, includes the customary quantity of eggs. In "Answers to correspondents," on page 94, in describing ice cream to "N. M.," the Journal says:

> For 1 quart of evaporated cream use 2 quarts of milk, then add 1½ pounds of sugar, stir, strain, and freeze. You may dilute the cream with 1 pint of water, but as this will make an inferior article we can not recommend it.

In this connection it may be stated that the trade name "evaporated cream" is simply a name for condensed milk. Therefore the ice cream which the Journal recommends to "N. M." is not at all like that which it described in the editorial article.

In the same journal for August, 1907, are given additional formulas for ice cream. In speaking of Neapolitan ice cream the following language is used:

> This is no special cream; it merely consists of 4 different flavors packed in layers into brick molds and cut into slices when served. The first layer being orange or lemon water ice, next strawberry ice cream, then chocolate, and lastly vanilla ice cream.

This is quite a different compound from the formula for Neapolitan ice cream previously referred to in this same journal.

In the same journal for September, 1907, page 101, is a description of "elk" ice cream, which is made as follows:

> Place 10 yolks of eggs into a farina boiler, add 2 vanilla beans, split in halves, set on a very slow fire, and beat the yolks until they form a thick body; remove the boiler from the fire and beat until cold. Now make Italian meringue of 4 whites of eggs and 9 ounces of sugar, add this to the beaten yolks, and when the composition is entirely cold, add 1 strong pint of whipped cream. When the composition is well mixed, add 8 ounces of preserved fruits cut into small dice and soaked in good maraschino, and last, 2 ounces of finely crushed macaroons.

It would be a little difficult if this were a puzzle to find the cream in the mixture.

The same journal, page 24, gives a recipe for maple ice cream. It April, 1907, in response to a query asking for the formula of New York ice cream, makes the following statement:

> There are almost as many formulas for New York ice cream as there are for plain vanilla ice cream, different makers having widely different notions as to the proper ingredients and method for New York ice cream.

Following this was a number of recipes for making a substance called "New York ice cream," each of them differing in essential particulars from the others.

The same journal, page 24, gives a recipe for maple ice cream. It is made of—

> 1 quart maple syrup, 1 pound granulated sugar, 12 eggs, 2 quarts sweet cream, 20 per cent vanilla. Boil sirup and sugar and pour in a thin stream over the beaten eggs, whisking briskly.

The editorial comment on the formula, which is furnished to Harris Brothers, Jamestown, N. Y., is as follows:

> It would seem that this mixture ought to make nearer 6 quarts than 4 (unless the machine is turned at slow speed) and still be very smooth and full bodied. The proportion of sweetening ingredients is abnormal. Cutting the sugar in half would improve the product. The milk fat contained is approximately 9.5 per cent.

It is evident that not only may ice cream, as commonly understood, be made of anything, but the journalistic advice is to swell it so as to increase its bulk. This is interesting inasmuch as ice cream is bought by measure and not by weight.

A "pure-food ice cream," the newest variety to which my attention has been called, is described in the June number of the Ice Cream Trade Journal, page 18. It is as follows:

> Sweet cream 5 quarts, cooked down to 1 gallon, being careful not to scorch it. It is cooled quickly in ice and stored until cold. This will raise the test of your cream and will give body to your ice cream. Formula is as follows:
>
> 4½ gallons, 20 per cent cream, 1 gallon condensed cream (your own make), 7½ pounds sugar, 3 ounces of best gelatin, 3 ounces of vanilla.

The maker of the recipe adds:

> This ought to make 10 gallons of as high-grade ice cream as it is possible to make for smoothness, good body, and elegant flavor. All ice cream requires something to make it smooth and keep it so. If a State prohibits gelatin, would it permit the use of rennet? They allow it to be used in cheese, so how could they object to it in ice cream? About 6 tablespoonfuls would answer.

The editor of the Journal makes the following comment:

> We can see no reason why anyone should object to the proper use of rennet in the manufacture of ice cream, but the fact remains that under a legal standard for ice cream that failed to mention rennet as an admissible ingredient its use could not be permitted.

The editor of the Ice Cream Trade Journal in the issue of August, 1907, page 24, makes the following statement:

> SILLY ACTION PROPOSED.
>
> From many different parts of the country come reports that some ice cream manufacturers are preparing to change the name of ice cream in order to comply with or to evade the law, as you please. If manufacturers are making ice cream there is no occasion to change its name; if what they are making is not ice cream then it is not ice cream that is to be given a new or a changed name.
>
> There should be no change in the name of ice cream. What there should be is the stiffest kind of a fight, even in the face of dire threats emanating from Washington and divers State food-control camps, to retain the name "ice cream" for every kind and quality of product justly entitled to bear it by reason of having borne it since the time when the name came into common use as the common name of a class or group of ices differing from that class or group known by the common name of water ices.

In the Ice Cream Trade Journal for September, 1907, in answer to the question "What is your best formula for French ice cream," the editor says:

> It is rather difficult to offer a best formula for French ice cream. Below we give two formulas, the second of which is similar to what is called Delmonico ice cream, except that the proportion of mix to finished product is greater.
>
> First formula, for 10 quarts: 24 whole eggs, 4 pounds sugar, 6 quarts cream, vanilla.
>
> Second formula, for 10 quarts: 3 quarts cream, 3 quarts milk, 2½ pounds sugar, 18 egg yolks, vanilla.

> Really the chief difference between French ice cream and an American cream containing eggs is that it is much more solid and somewhat smoother because of its closer grain, and this is due to its being frozen in such manner that there is not much increase in bulk.

The Ice Cream Trade Journal of October, 1907, contains a number of English formulas, submitted for the purpose of showing that English cream ice and American ice cream are similar products. Then follow 7 recipes for making different kinds of ice cream. All of these recipes contain eggs, but none of them gelatin or any other stiffener.

A small pamphlet entitled "Hand Book on Ice Cream," by Adolph Kramer, published by the Sioux Publishing Company and received at the Department of Agriculture on July 5, 1907, gives interesting information from the trade standpoint. The pamphlet is only 12 pages of a single column each, and is sold for $5 a copy. On the front page occurs the following statement:

> This little booklet tells you how to manufacture a prime ice cream at 10 cents per gallon equal to a full cream and perfectly healthful; formulas for fancy creams, fruit ices, fruit frosts, sherbets, whipped cream, etc. This booklet is worth $100 to you.

On page 3 occurs the following language:

> Ice cream at 10 cents per gallon sounds good to you, doesn't it? Of course it does. * * * Ice cream has been made and used in this country for over one hundred and ten years and it has increased in popularity every day since and will continue to do so. The Italians claim the honor of first presenting ice in solid form, and for that reason it is presumable that the name "Neapolitan" as applied to ice cream will never become obsolete. The name "Neapolitan" is applied to custard cream in general. It is also used to designate a fancy cream. The day for using a straight cream, testing from 25 per cent to 30 per cent of butter fat, has gone by and should go by, though some manufacturers claim they are using 20 per cent butter fat test and producing all cream and that their trade is constantly increasing. However that may be a full cream is too rich for the ordinary person's stomach. Manufacturers should aim to produce an ice cream that any person with a weak stomach should be able to eat all he wants without fear of being made sick, and such a cream is just as pleasing to the taste and just as healthful and far more satisfactory than a straight cream, not taking into consideration the extra profit for the manufacturer. The author has analyzed a large number of the preparations on the market which is used to improve and lessen the cost of ice cream, and when you have read this little booklet through you will be able to use your own preparations without paying someone else 1,000 per cent profit, like some of them on the market. Dextrine! Dextrine! Dextrine flour is one of the principal ingredients that does the trick. Now, this article is perfectly healthful and will give good satisfaction. It doubles the quantity, saves one-half the labor, saves one half of the ice, and saves one-half of the salt, it keeps them twice as long, it will not separate, and will not ice. By its use whipped cream can be made of 25 per cent cream in half the time and stand twice as long as 50 per cent without it. It will make good ice cream from pure milk.

Then follow directions for making all forms of cream. On page 9 is the formula for ice cream at 10 cents per gallon, which is as follows:

> It is possible to produce a good ice cream for 10 cents, figuring milk as 14 cents per gallon. Here is the formula:
>
> Powdered gelatin 7 pounds, dextrine flour 3 pounds, mix thoroughly. * * * A powdered gelatin good enough for this may be bought for 20 cents per pound at the factory, and dextrine flour for 3 cents per pound. Dextrine may be bought from any glucose refinery.

One pound of the above mixture at 15 cents and 5 gallons of milk at 14 cents a gallon are the directions given for the final process.

The evidence cited shows that the products which have been sold as "ice cream" for many years in this country may be of almost any possible composition. We have found recommended for use in its composition, milk, skimmed milk, condensed milk, evaporated milk, vegetable gums, starch, dextrine, flour, eggs, gelatin, and other substances. A formula has been offered for making ice cream that costs not more than 10 cents per gallon. It is evident that many of these substances are used simply because they are cheap and add bulk to the mixture and without any regard to their relations to health and digestion. So-called ice cream, having a definite name, it has been shown by the trade journals, is made up according to a half dozen formulas, so that it is impossible to state what an ice cream called by a definite name is. There is no uniformity followed in its manufacture, the sole object seeming to be to make it as cheap as possible and still secure a market therefor. It is evident from the authorities that the consumer is not given any kind of information at all when he purchases a substance known as ice cream, except perhaps that it is frozen. Even this does not seem at all times to be necessary, since ice cream has been offered and guaranteed to stand up for hours after it has been removed from the freezing machine.

Having given in the preceding pages a summary of the authorities respecting the composition of ice cream, it is possible now to have a clear vision of the significance of this term in commerce before the advent of the food and drugs act. A study of the data will show in the first place that the frozen custard, which is said to have had its origin at Naples and which in this country has been known as Neapolitan ice cream, never was known in the country of its origin as ice cream, but by other terms entirely different in signification. In general, it may be said that the term ice cream is not used in any of the European countries, nor has it ever been used with the possible exception of its occurrence of late years in English menus, due to the crowd of Americans who visit England every year, especially during the summer season. The claim therefore that any kind of a pudding, or mixture of any description, has from the first been called ice cream does not appear to be sustained by the evidence.

In regard to the American custom, it appears plainly from the authorities quoted that there has always been made in this country a genuine ice cream composed solely of rich cream, sugar and harmless flavor, and this substance has been recognized and sold as ice cream from the very first. It has very often been designated in this country as Philadelphia ice cream, and this prefix indicated, at least to the trade, the character of the goods. The term "Philadelphia ice cream," however, would carry no meaning to the consumer except one of a geographical signification. The claim therefore which has been made that real ice cream has not always been made and sold in the United States does not appear to be verified by the authorities which have been cited. It is only fair to presume that Philadelphia is not the only town in which such ice cream has been made, but that it has been made very generally in all parts of the country. Hence it appears as established beyond any reasonable doubt that a real and genuine ice cream has always been an article of commerce.

The claim that has been made that the people do not want genuine ice cream must be considered from two points of view. If by this is meant that the people in general want an ice cream as cheap as it can be bought, then the claim may be regarded as a fact. If, on the other hand, it is meant that consumers do not like the taste of genuine ice cream, there seems to be no evidence whatever in the way of its verification. Experience has shown that not only do the people, as a rule, like genuine ice cream, but they prefer it to any kind of frozen custard which may masquerade under the name of ice cream. The claim which has been made that genuine ice cream is not wholesome also lacks any kind of evidence. The fact that physicians prescribe genuine ice cream for invalids is an indication that it is regarded by the medical profession as a wholesome article of diet. It is undoubtedly true that on account of its richness in butter fat genuine ice cream is an article of diet which should be consumed in some moderation, not only by the sick but also by the well. There is no evidence whatever to show that genuine ice cream is unwholesome in any degree except it may be contraindicated in certain diseased conditions of the stomach or digestive organs, or may be eaten in excessive quantities. These facts, however, can not in any sense be cited as evidence of unwholesomeness. On the contrary, it may be said with full assurance of verification that the average consumer prefers the genuine ice cream to any of the mixtures which may be substituted therefor. It is recommended by its organoleptic properties as superior to the mixtures containing various added substances, used chiefly to give bulk or firmness to the mass. From the point of view of the general consumer the genuine ice cream is to be preferred for palatability to any of its substitutes.

ICE CREAM STANDARD.

The standard for ice cream was suggested by the committee on standards after a long and careful study of the composition of ice cream, and the general character thereof, the meaning of the term, and the desirability of having it under the food law express some definite meaning. The form in which it was finally established is found in circular No. 19, of the Office of the Secretary of Agriculture, issued June 26, 1906, page 7. The standards read as follows:

> 1. Ice cream is a frozen product made from cream and sugar, with or without a natural flavoring, and contains not less than 14 per cent of milk fat.
>
> 2. Fruit ice cream is a frozen product made from cream, sugar, and sound, clean, mature fruits, and contains not less than 12 per cent of milk fat.
>
> 3. Nut ice cream is a frozen product made from cream, sugar, and sound, nonrancid nuts, and contains not less than 12 per cent of milk fat.

No standards were made for other varieties of ice cream.

Before these standards were issued full opportunity was given to the trade to discuss the tentative standards which had been proposed and on which criticism and advice were asked. All this evidence was considered carefully by the committee before the final publication was authorized by the Secretary of Agriculture. Some of it was favorable to the creation of a standard and some opposed thereto. In order that the subject may be fairly presented, excerpts from this evidence are submitted. I give first the remarks made before the standards committee at its meeting in Louisville in December, 1906, by Mr. Samuel R. Kennedy, Pennsylvania, and then of others representing different views:

> Ice cream was originally invented by Florin in the city of Naples in sunny Italy about a century and a half ago and to-day it is still made and sold in Florin's café by his lineal descendants. It was composed of honey, fresh eggs, and sweet cream, which was frozen in long cylindrical shapes of various colors and served in a wine glass. * * *
>
> The agitation produced by the passage of the pure-food law, establishing a standard for ice cream has occasioned greater interest than anything heretofore known to the trade and all with one accord have begun to query and question "where are we at," and what will we do for a thickener and what formula or "mix" shall we adopt to comply with the new law which goes into effect upon January 1, 1907.
>
> The law defines ice cream to be composed of cream, sugar, flavor, nuts, and fruit; and the commission created under the law has set the standard at 14 per cent butter fats for vanilla and chocolate ice cream and 12 per cent for fruits and nut ice cream.
>
> It has become the custom for several years past for the trade to use gelatin and refined glue under various proprietary names to "body up," and "thicken" and adulterate for the purpose, ostensibly of improving the product, with anywhere from 1½ ounces to 7 ounces of this product of the vat from abattoirs, consisting of horns, hoofs, pieces and scraps of skin, hides, shin bones, and

other unwholesome matter from our own and South American slaughterhouses. The respectable manufacturers or makers of ice cream did not use gelatin or other thickeners for the purpose of cheapening their product, but for the purpose of producing the velvet smoothness so much admired, and also for the purpose of insulating the frozen watery or aqueous portion of the cream and preventing it from swimming or turning into "soup," upon the slightest exposure. To avoid this dilemma, the cream had in the past been reenforced or "bodied up" by making custard (hot or cold), the old and ancient practice of adding fresh eggs and heating gently to form a custard or by adding cornstarch, arrowroot, potato starch, gum arabic, or tragacanth, ground gelatin, tapioca, etc., some of these not requiring heat to form a mucilaginous body in the cream, aided in keeping the ice cream firm.

The use of same is now prohibited under the new law under a penalty that will prevent even the lawless from risking its execution. There is some hardship to the commercial ice cream in the double standard. Ice cream as ordinarily made is run up in 10-gallon batches from 5½ to 6 gallons "mixes," and after being "doubled" and "frozen," and then flavored, and rerun long enough to diffuse the fruit, extracts, nuts, coloring, etc., evenly and uniformly throughout the mass, it is then packed in "packers," or suitable tins to suit the customers' orders and hardened ready for sale, shipment, and delivery.

Now, by this method it is quite impracticable to the ordinary manufacturer to make exactly the quality of the two standards, for instance, lemon and vanilla are flavored almost identically by the same quantity of extract, but the one under the law may contain 12 per cent butter fat and the other must contain 14 per cent, while chocolate ice cream, which is enriched by at least 1 pound of cocoa butter fat, can be the same as vanilla.

I would therefore respectfully ask the committee upon food standards to make a uniform standard of the lower quantity named, viz, 12 per cent and require all ice cream to be made of that quality or above it.

In making "runs" of "fruit" ice creams it is the practice to dip off a gallon of cream after being "run up" or "doubled" and fill in a gallon of crushed or macerated fruit, and thus the standard is involved, and there is a risk of an honest manufacturer disturbing the percentage, as ice cream in the soft state is difficult to measure with a dipper.

Next the variation of the cream supplied to the manufacturer is beyond his control and would necessitate his calling in the chemist as a daily assistant to keep the milk dealer and creamery man up to his contract.

The reputation of ice cream as a delicacy and a food for the sick was not achieved by the large manufacturers who now are the largest "calamity howlers" over the hardships of the new law, but by the small confectioners who made a neighborhood reputation, * * * but as ice cream became more and more popular, the machinery supply man began to manufacture machines for the trade, the long cherished secrets and formulas slipped into other hands and books of recipes were published. Then came the steam ice cream factory with its dirt and slop—dark, damp, noisome, underground, or above ground, in some stable or shed—the whirring and buzzing work went on and fierce competition drove down prices, and along came the "devil with his glue bags," tempting with his arguments, a "bigger yield," "less ice," "more velvety smoothness," "fast runs," "saving coal and ice," "nonmelting quality," use "more milk" "less cream," until after while "glue and water" began to play an important part in the largest establishments run by steam and electricity.

There has grown into general use the last thirty or more years among the more respectable ice cream makers, the addition of a gallon of so-called heavy evaporated cream or plain superheated condensed milk (unsweetened) to each 4 or 5 gallons of 20 to 22 per cent cream.

This was a pure whole milk concentrated "in vacuo" to about three to four times the thickness of the richest milk and served as a thickener without being foreign to the dairy or the cow; this avoided the necessity for using thickeners, starches, cornstarch, potato starch, arrowroot starch, gums, gum tragacanth, senegal, Arabic, etc., and made a beautiful smooth velvet-like product and double as much as cream alone would do. It had the advantage of purity, wholesomeness, digestibility, and cost about the same as cream. It would prevent the cream from swimming when dished up, or when transported long distances to customers who lived out of town in the summer time; but this formula was only used by the best family ice cream purveyors, as "glue was cheaper;" the "lordly mushroom" compounders could not afford to drop the large doses of water glue that enabled them to work up into "the only absolutely pure ice cream."

The term cream should be also understood under the new pure-food law by the ice cream trade.

The standard for cream calls for not less than 18 per cent of butter fat, and it is liberal in several respects, as it does not designate hand-skimmed cream, pasteurized cream, separator cream, centrifugal cream solidified cream, or evaporated cream, if they come up to the standard of not less than 18 per cent butter fat. This will be a great help to the ice-cream solidified cream, or evaporated cream, if they come up to the standard of not less than 18 per cent butter fat. This will be a great help to the ice-cream maker, for so long as he uses this or a higher standard he will be sure of coming up to the standard required by law. Some provisions should be provided under the law to suppress some grades of frozen mixtures now upon the market posing as cheap ice cream which do not contain cream, evaporated cream, whole milk, or a trace thereof, and which are sold to children who have only a few pennies to spend and want as much for their little sum as possible. While I recognize that frozen custard, frozen junket, and frozen jellies can be made clean and wholesome, I think it is but right that poor men's children should be safeguarded, and I hope that the pure-food commission and the committee upon food standards will make a low standard as well as a higher one and will interest the Department in the subject so that the popular cheap frozen products within the reach of the humblest citizen may be safeguarded by the august eye of the law and be subject to the intelligent scrutiny of the chemical inspection of the Department.

It is my opinion that all the trade are desirous of living up willingly to the standards, and in fact they see the beginning of better trade conditions and higher prices as a result. While it has placed them in a quandary as to how to proceed in the premises, as the instructions thus far have been quite meager, nevertheless I am confident they will be glad to accept the new standard. It would however materially assist if the Department would in due course issue a bulletin which would give instructions as far as deemed advisable by your Department.

CRITICISM OF E. G. ECKERT AND OTHERS.

Dr. E. G. Eckert, Secretary of the Ice Cream Manufacturers' Association of Pennsylvania, made the following statement at the national convention of ice-cream makers held in Chicago in February, 1907. The account is taken from the Ice Cream Trade Journal of February-March, 1907:

> Pennsylvania is the Keystone State and seems to take the initiative in most things political. We do not have a Matthew Stanley Quay any longer, but we have some people who have learned politics from Matthew Stanley * * *. We are free-born citizens and we do not propose to have any commissioner of agriculture or his coterie of associates tell us what to do as against that which has been done for one hundred and fifty years. We had with us at Harrisburg Senator Tustin, chairman of this committee, who assured us that he recognized that there was an injustice being done to a manufacturing industry which ought not to be tolerated. We are untiring in our efforts to bring about the passage of a pure-food law which will benefit the public without injuring manufacturers and dealers. We do not want a law that makes standards. I am not in favor of any standard. When you ask for 1 per cent or 2 per cent or 8 per cent of butter fat in ice cream, you are asking for an arbitrary standard. We are Americans. Our standard is as high as the heavens, and whatever people want and are willing to pay for give them. * * * You must get your State association organized and prevent the insertion in the agricultural bill in the Senate of a provision for standards.

Mr. Thos. E. Lannen, a lawyer, also addressed the convention on the subject of standards. During the address he said:

> "My only suggestion at the present time on this standard for ice cream would be to adopt a standard which will permit you to conduct your business as you have been doing it in the past, and if there is any practice going on in your industry which is illegal and which in the minds of the majority of the men present here to-day should not be permitted then you should draft such a standard as will stamp out that practice."

Mr. N. Lowenstein, Secretary of the Sethness Company, of Chicago, in the course of his address respecting the standards for ice cream, said:

> "In answer to a telegram which I sent to the two Senators from Illinois I have a reply from one of them, reading as follows: 'Your telegram of recent date is at hand, and contents noted. Your suggestion relating to the proposed provision regarding food standards in the agricultural bill shall receive due consideration I can assure you.'"

It may be said in passing that the ice cream makers were not the only persons who endeavored to have the authority to fix food standards abolished. There are many other manufacturing interests which object to any standard whatever being set for their products. The standard, however, for ice cream to which objection was made was established long before the authority to establish standards was withdrawn. Continuing, Mr. Lowenstein said, speaking of the authority to fix standards:

> "Certain interests endeavored to have this same provision inserted in the agricultural appropriation bill of 1904, 1905, 1906, and again this year, and in each instance it was ruled out on a point of order as irregular legislation.

Efforts were made to have this objectionable provision reinserted in the Senate bill and a great many food manufacturers and organizations immediately communicated with their Senators requesting that no one be given arbitrary power to fix food standards under the agricultural appropriation bill."

Mr. Jackson, of Sterling, Ill., said in the same Journal:

My formula consists of milk, cream, condensed milk, and gelatin, and I worked that out by days of experimenting. * * * The result is I manufacture 40,000 gallons of ice cream every year and I never have a complaint. I ship it from Sterling up to Dixon and to Freeport and over to Galena and down to El Paso, even down to Wheaton, a suburb of Chicago. One of my customers is the best drug store in Wheaton. The smallest children eat our ice cream in quantities. My youngest child was fed ice cream before he was through nursing. He is three years old now and gets from 2 to 3 dishes of it in hot weather, 4 or 5 if he wants them. The doctor's bill for my entire family is not over $5 a year.

Extracts from the remarks of Mr. Chisholm read as follows:

I believe in giving the people what they want. We have in our place gentlemen who make what they call a pure cream, cream that they do not use any gelatin or anything of the kind in. We have held customers against men who claim to make a pure cream of 14 per cent without any gelatin. People demand a cream that is not so rich as 14 per cent. I would like to speak a word in regard to what the gentleman from Sterling said about the question of cream. He seems to think that if we adopt that 14 per cent standard we shall decrease the amount of ice cream used. We very likely would. Suppose we decreased it 25 per cent we would still have to increase the amount of cream we use in order to make that amount of ice cream, according to my way of figuring. Where are we going to get that cream? It will not put the cream back to the creamery; we shall have to put up the price more than 20 per cent over what the creamery now pays, as he says he has been doing. In order to get the cream we shall have to take it away from the creamery and pure butter will go up. Suppose it does not decrease the amount of ice cream that we sell 25 per cent. If we are now making, say 7 per cent, take that as an illustration, if we go to making 14 per cent it takes twice as much cream as it does now. Where are we going to get it? It not only increases the price of cream we sell by the value of the extra cream used but all the cream we have to buy will cost us more money and we shall have to increase our price more than in proportion to the increase in butter fat.

Mr. Woodhull called attention to the fight the ice cream makers are making against the standards. He said:

We sent out some telegrams to-day that we would like to have ratified and made official by the association and spread on the record. We took it upon ourselves to send these telegrams, knowing that they should have been sent as soon as possible so we would not have to wait until evening. We have a telegram to the Hon. E. D. Crumpacker, who so brilliantly, earnestly, and successfully took a stand in the House against irregular legislation being incorporated in the agricultural appropriation bill:

"The Hon. E. D. CRUMPACKER,

"*Washington, D. C.*

"The National Association of Ice Cream Manufacturers, in convention assembled, desires to thank you for your inestimable services through which you brought about the exclusion of the irregular legislation in the House appropriation bill, thereby preventing the one-man power from destroying their industry.

"J. H. FRANK, *President.*"

President FRANK: Gentlemen, Mr. Crumpacker is a Congressman from Indiana and he will do the right thing. Are you ready for the question?

The motion was then put and unanimously adopted. Another telegram was sent to

"Senator PROCTOR,

"*Chairman Committee on Agriculture,*

"*Senate Chamber, Washington, D. C.*

"The Illinois Association of Ice Cream Manufacturers, in convention assembled, earnestly requests that you do not allow reinstatement in agricultural appropriation bill of parts stricken out in the House from bureau of chemistry section.

(Signed) "ILLINOIS ASSOCIATION OF ICE CREAM MANUFACTURERS,
"R. A. WOODHULL, *President.*"

Mr. McCREA said:

"The man who goes out into the field to compete for business and sells frozen water for ice cream is not a success, and you know it as well as I know it."

President FRANK:

"They come pretty near doing it sometimes."

After much discussion a rising vote was taken as to whether the convention should recommend to the commission a 14 per cent butter-fat standard, an 8 per cent butter-fat standard, or no standard at all, and the last proposition was carried by an overwhelming majority.

The above quotations from the proceedings of the Ice Cream Convention are given to show that the product which is sold as ice cream, or at least was sold as ice cream before the enactment of the food law, has no definite composition. No one can have any idea, as is shown by the statements made by the makers themselves, what the substance purchased as ice cream really is. As the President of the Association very aptly remarked, "Some of the members evidently were selling frozen water as ice cream, or nearly so."

In The Ice Cream Trade Journal, October, 1906, page 23, there is an editorial article on "Butter Fat in Fine Ice Cream." This article states that the late Charles Ranhofer, who was for many years chef of Delmonico's, in his work entitled "The Epicurean," published in 1894, devotes 50 pages to ice creams and ices. In quantity of butter fat the plain creams which he describes are as a rule richer than his fancy creams. The editor says:

> Under the head of "Vanilla Ice Creams" you will find instructions for making 9 kinds. One, a fancy ice cream, contains no milk or cream whatever. One is similar to New York ice cream, or frozen custard, and has a butter-fat content of about 2 per cent. Four others roughly calculated are well under 10 per cent of butter fat. One shows 11 per cent, one 14, and one 17. * * * We find that the butter-fat content is low in the majority of ice creams. True a few formulas show a high percentage of butter fat in the mixture, but, on the other hand, we find a number of formulas for ice cream that do not call for any cream and we have drawn attention to one that leaves out milk as well as cream. It is evident that a quality standard for ice cream specifying a minimum butter-fat content, unless that minimum is low, would prevent the sale of many fancy frozen dainties that were sold as ice cream before hokey pokey was invented. * * * For anyone to say that the term "ice cream" covers less to-day than it covered fifty years ago is absurd; therefore a standard that requires 6 out of 10 ice creams to be sold under another name is absurd. There's an old saying—not wholly untrue—that the law is an ass, but is it necessary in order to prevent fraud for those charged with the enforcement of the law to be absurd? Ice cream is a compound in which (except in rare cases) the principal ingredients are milk products, but if one reduces the butter-fat constituent in his compound or eliminates it and substitutes something equally wholesome and nourishing who shall say that he has not made ice cream as good as or even better than ice cream containing a specified percentage of butter fat? While we do not believe that a standard specifying the butter-fat content in ice cream is necessary to prevent fraud the establishment of a reasonable standard would prevent the sale of cheap frozen compounds unless they were plainly labeled to indicate their character, and this we believe would necessarily preclude their being served in individual portions, as in restaurants and at soda fountains. But what is a reasonable standard? Certainly not a standard that fixes the minimum butter fat content above 8 per cent nor a standard that does not admit of the substitution of fresh eggs for butter fat pound for pound.

The Kymo Company, manufacturers of food preparations, of Little Falls, N. Y., under date of February 25, 1907, submitted a protest against the standards for ice cream. The reasons for demanding a change are as follows:

> Inclosed, we hand you an amendment to the national definition for ice cream as given in circular No. 19. To our amendment we have appended an argument setting forth briefly numerous reasons why the present national definition should be repealed or amended to agree with our definition. You will find a recapitulation of our reasons on the last two pages of the inclosed argument.
>
> Because the States show a disposition to adopt the national standards we deem it very important that these be as nearly right and just as it is possible to make them. If they are not just and right those States that accept them will be led into errors that will in some cases result in hardships to its citizens. On the other hand, those States that refuse to accept the faulty standards will not be in full accord with those that do, nor with the National Government in the very important work of suppressing the traffic in adulterated and harmful foods and drugs.
>
> If the Agricultural Department or those in control of the matter of standards insist upon unreasonable standards like that for ice cream, will not the public

generally, especially the manufacturing element, combine with more sordid interests to bring about the abridgment or extinguishment of the power and authority to make standards?

Believing that the national definition for ice cream is very faulty and will work untold hardships to manufacturers and consumers alike, especially if adopted by the States, we respectfully submit our definition and argument.

The definition proposed by the Kymo Company is as follows:

Ice cream.—Ice cream is a frozen product made from cream or milk, fresh or condensed, and sugar, with or without a natural flavoring and with or without the addition of other harmless vegetable and animal ingredients or products.

The company also says:

The term ice cream as now used is not a misnomer nor is it misunderstood by the consumer. This name is established in the minds of the manufacturer and consumer alike as that of an article that is made from recipes or formulas that vary greatly as to their ingredients. The consumer thus makes or purchases ice cream of a kind or quality that accords with his taste or means. There is no evidence of dissatisfaction on his part with the present popular definition or with the present product, therefore there is no cause for a new definition or for legislation along this line on the grounds that the public is being deceived or imposed upon by the sale of adulterated or misbranded ice cream.

The term ice cream, in the minds of the consumer and the manufacturer, does not indicate that the frozen product is made from any particular amount or proportion of milk fat. To those who are conversant with the art of manufacturing ice cream, including the confectioner, baker, caterer, and housewife, the name suggests a variety of ingredients, and the quality of the article is not based on the amount of milk fat contained. * * *

From the foregoing, it is obvious that to protect the public it is not necessary to restrict the term "ice cream" to frozen cream, sugar, and flavoring, as this is not the popular definition and is not what the consumer makes when he manufactures his own product. To the consumer and manufacturer alike ice cream made according to the Agricultural Department's definition is a new product under an old and familiar name.

We believe that most doctors will agree that 14 per cent of milk fat in ice cream is a larger proportion than is good for the average individual. This is particularly true during warm weather, when ice cream is consumed most liberally. As regards healthfulness, whether taken as a food or as a cooling confection or delicacy, we believe that a pure milk ice cream is preferable to one made from pure cream, just as much so as milk is better than cream for the average individual to drink.

The Kymo Company also makes the following statement regarding the determination of the percentage of fat:

In all that we have seen or heard on the subject of how to figure the percentage of fat in ice cream the basis has been the relation of the milk fat to the entire weight of the raw materials. On this basis it has been estimated that cream testing 17 per cent will produce ice cream showing 14 per cent of milk fat. If this figuring is accepted ice cream made in a slow speed power freezer will cost nearly double as much as that made from the same materials in a freezer that whips the materials into double its original volume. Does this not look like discrimination in favor of the man with the high speed freezer?

The company also makes the following observations upon the very common practice at the present day of practically doubling the volume of ice cream. As ice cream is chiefly sold by volume it is evident that any process which will make out of a given amount of materials double the volume must be a source of profit to the manufacturer. Just what benefit this expansion of volume is to the consumer does not plainly appear:

> As is well known, most wholesale manufacturers make 40 quarts of ice cream from 20 quarts of materials. What is to prevent the manufacturer from still further diluting with air his 17 per cent milk fat cream if the test for the milk fat is on the basis of the weight or volume of the raw materials or of the melted product?
>
> If we must have a milk-fat standard let it be one that will result in uniformity in the finished product under all processes of manufacturing and that will not give the man with the fastest freezer a practical monopoly. If the Department must have a standard let it be one that will not tempt the manufacturer to neutralize the increased cost of his raw materials by increased expansion.

The force of the above argument is not apparent. Inasmuch as the percentage, unless otherwise stated, is always a percentage on weight it does not make any difference in the estimation of the percentage whether the materials have been expanded to 2, 3, 4, or 5 volumes. The relative weight of fat to the materials is not changed by the process of expansion, since the air which is used in the expansion is practically so light as to add nothing of any consequence to the weight of the expanded article.

Great stress is laid by the Kymo Company upon the fact that ice cream which contains 14 per cent butter fat is a new product not known hitherto to the trade. The company says:

> In view of the facts related in the foregoing, we suggest that if the Government or the Agricultural Department requires a name for the product of its definition it either select a specific name that will not interfere with established trade conditions or let the term ice cream apply generically as it does to other frozen confections. As a specific name for the Department's new product we might suggest the following: "Pure cream ice cream, cream ice cream, cream ice, iced cream, or frozen cream." The term cream, however, would not have the same significance in the Department's 3 definitions because of the variation in the milk fat in those definitions.

Apparently a more just construction of the requirements would be to require a definite name for the variations instead of for the pure article, thus introducing the names "Milk ice cream, skimmed milk ice cream, condensed milk ice cream, evaporated milk ice cream, gelatin ice cream, egg ice cream, coal tar dye ice cream," etc.

Summarizing, the company closes its remarks as follows:

> We believe that we have shown conclusively
>
> First. That there is no need of a new definition or standard for ice cream.

Second. That ice cream as made by present processes is noninjurious to health and satisfactory in quality to the consumer.

Third. That the manufacturer and consumer are at one in their respective understandings of the term as now used.

Fourth. That the present process of manufacturing, including the formulæ, accords with the accepted meaning of the term in our standard dictionaries, also with its history.

Fifth. That it is not misbranding to apply the term ice cream to the usual frozen products by that name.

Sixth. That cream is not a specific term used only as a name for the fatty part of milk, this being one of several applications of the word.

Seventh. That Article f of Regulation 12, Section 8, does not apply to ice cream, as the name of this article is not derived from one of its constituents but from its own qualities as a product.

Eighth. That the process to which the Agricultural Department proposes to apply the term is not the ice cream of commerce or the home, but a new product.

Ninth. That no definition fixing a standard of milk fat is practicable or desirable.

Tenth. That the term ice cream has become a valuable trade name, the establishment of which, by advertising and other means, has required the expenditure of large sums.

Eleventh. That, without good cause, the Government has no right to condemn the term ice cream for application exclusively to another product.

Twelfth. That, as a model for general acceptance by the States, the Department's definition should be amended in accordance with our definition.

Under the heading "Trade Customs," the Lancet makes some very proper comments as to the dishonest practices of which many vendors and manufacturers are guilty under the convenient designation of "trade customs." Our contemporary observes that the term "trade customs" in some quarters appears to be the modern synonym for malpractices.

So many defendants shelter themselves, or attempt to shelter themselves, behind the plea of trade custom that it would be interesting to have a list of "trade customs" published.

The public have a right to know what trade customs are. We doubt very much if the public know quite as much about them as the trade. Police court proceedings enlighten us considerably at times, but there are so many "trade customs" that we plead for a glossary of them. We fancy that we should be fairly safe in saying that such a compilation would open our eyes to a string of petty practices designed more or less to cheat the purchasing public; trade customs in fact, which, though approved by the trade, are, strictly speaking, illegal transactions. We should like to see appointed a royal commission on "trade customs." The selection of the commissioners, who, of course, would be authorities on the subject, would be interesting, and the evidence of the witnesses would at least be amusing if not instructive. The final report would have the word "swindle" written in every one of its conclusions—that is to say if the commissioners honestly set about their business. These may be strong words, but day by day we read in police court proceedings how indictment after indictment is met by the sickening excuse of "trade customs." Brown paper is found in the soles of boots; it is a trade custom. Silk containing cotton is sold as pure silk: it is a common practice of the trade and therefore a justifiable one because the trade recognizes it. It is also at times the trade custom to call an article brandy which is not brandy, soda water which is not soda water, butter which is not butter, and so on ad infinitum. In fine, it will be found that "trade customs," as a rule, do not call a spade a spade and things are not what they seem. The term "trade customs" is a cloak, is not in many instances honest, and in an equal number of instances exists to evade the law. The law should recognize no trade custom which is not straight dealing.

The British Food Journal has repeatedly called attention to this matter and has indicated the absurdity of the "trade custom" excuse. Some of the most insidious forms of swindling are recognized and practiced by certain trades under the description of trade customs upon which the light of the police court never shines. Those firms who are guilty of such malpractices can well afford to take the remote chance of being found out and of having to pay a small fine because they find their course of procedure exceedingly remunerative. There seems to be an ingrained desire in certain individuals to cheat their neighbors and compete by fraud.

The Horton Ice Cream Company has made the following representations respecting the standard:

Ice cream is a frozen product made from cream and sugar, with or without a natural flavor, and contains not less than 14 per cent of butter fat.

This is the official definition of ice cream according to U. S. Circular No 19, Department of Agriculture.

If the above is a correct definition of ice cream then for the past fifty years and over there has been little or none made and sold.

Cookbooks dating from 1853 do not describe ice cream in this way and it has not been according to American custom to make it in this manner.

If the authorities will consult the leading and standard cookbooks published in this country they will find various ways of making ice cream, and why should a law be made where there can be only one way of making it and then only a product showing 14 per cent.

If this standard should be adopted by this State and the United States it would fail to bring about the desired effect for the reason that ice cream has not been made to show 14 per cent generally, and instead of dealers endeavoring to comply with the standard ice cream, or what used to be called ice cream, would be sold under a new name and in time the term would become obsolete. It is no guesswork but a fact that the dealer who attempted to sell standard 14 per cent goods would not be able to compete with the man who sells what was formerly known as ice cream under a new name, either in price or quality, and the practical ice cream man knows it.

There is a market for a frozen product showing less than 14 per cent butter fat made with or without eggs, and with or without gelatin, and with or without condensed milk, and with or without flavor, and time will show it, for to-day the leading hotels make an ice cream with eggs and they will not discontinue making this product should they be obliged to change the name, and it will be found that this style of ice cream is made by the best men in the business and the per cent of butter fat would be found to be about 8 per cent.

Make a liberal interpretation of the law, say "Ice cream is a frozen product made of cream and sugar, with or without milk, condensed milk, gelatin, flavor, or eggs, and contains not less than 8 per cent of butter fat," and dealers will use their best endeavors to live up to it and see that others do, but if the proposed standard be adopted dealers can make such fine goods under a different name that are equally if not more delicious that ice cream in not many years would only be a name.

These standard cookbooks are not nor were they published to instruct manufacturers how to make their goods, but that housewives might know how to make the best of everything, and not with a view of seeing how cheap everything could be prepared to put before their families.

Gelatin is just as important an ingredient of ice cream as sugar, for without it ice cream could not be sold commercially for the reason that it would get icy and not fit to use.

A very interesting chapter on ice cream is contained in "The Epicurean," by Chas. Ranhofer, chef of Delmonicos, previously quoted. In the preface the author says:

In publishing this work I have endeavored to fill a much-needed want, namely, the best and most effectual manner of preparing healthy and nutritious food.

This edition contains innumerable recipes which I have simplified and explained in a comprehensive manner so as to best meet the wants of all. It suggests, also, many useful and important hints to those about entering the profession.

Recipe 3451 describes fresh-fruit ice creams which are to be made without eggs or cooking. The mixture which is used for the process is composed of 3 pints of cream, a pint of milk and a quart of the juice of the fruit. Peach ice cream is described as made with two-thirds of cream and one-third of the fruit pulp.

The most important point which is brought out by Mr. Ranhofer is the fact that he never uses the words "ice cream" alone to represent any of the mixtures which are usually sold under that name. I will quote some of the terms which he uses:

Ice cream al la Cialdini; Andalusian ice cream chocolate and cocoa ice cream; cinnamon, ginger, or pumpernickel rye bread ice cream; fresh fruit ice cream; nougat ice cream or nougat Nepolitan cream; pistachio ice cream; burnt almond ice cream and with angelica; rice ice cream; rice ice cream with citron, garnished with truffles; Italian meringue; virgin cream with orange flower water and noyau; ice cream with almonds; ice cream with eggs and black coffee; ice cream with roasted or boiled chestnuts, etc.

In all these mixtures into which any extraneous bodies are added Mr. Ranhofer is careful to give the name so as to distinguish it from the plain term of ice cream. Thus no false idea is conveyed to the purchaser respecting its quality or composition.

THE QUANTITY OF BUTTER FAT IN ICE CREAM.

The data which have been cited indicate that there is no tendency in the trade to secure any uniform quantity or standard of butter fat in ice creams. The authorities show that an ice cream may have from a mere trace of butter fat up to 17 or 20 per cent. The consumer, therefore, has no indication in buying a so called ice cream of the quantity of cream or butter fat which he is about to secure, nor would a physician in ordering ice cream for a patient have any information of the character of the food that the patient was going to eat; assuming that he is getting a genuine ice cream, he may be giving an invalid a lot of wholly indigestible materials which his stomach in its weakened condition would be utterly unable to digest.

The claim that the manufacture of genuine ice cream will make it too expensive for common use does not seem to be based on any reliable data. That real cream sells for more than an imitation and that it should sell for more no one will deny. If a man buys two volumes of a mixture containing 8 per cent of butter fat as ice cream, he may pay no more for it than a man who buys one volume of real ice cream. The answer to the question of increased cost would very properly be diminished volume. It would surely be advantageous to the consumer if he put into his stomach a less volume of the frozen mixture than he usually does when he buys an ice cream of commerce in which water is the chief constituent.

The claim that the dairies of the country would be unable to furnish cream for making genuine ice cream is wholly unfounded. The dairies of the country are interested as well as the sanitarians in having ice cream pure and true to the name. They will be able to supply the legitimate demand for the cream of which the article is made.

The protests against the standard for butter fat fixed by the Secretary of Agriculture under authority of Congress, in so far as the briefs and arguments which have been offered are concerned, seem to be wholly without merit. The same protests were made against fixing a legal standard of fat in milk, against the elimination of the quantity of water in butter, against the requirements for purity of almost every food product. Whenever an attempt is made to fix a standard of purity for a food product, all the people who are engaged in making a debased article of that kind enter the same kind of a plea. There seems to be no basis for a protest of this kind. There is no ethical or legal reason why the purchaser of ice cream should not have some definite idea of what he is getting. The conditions which obtained before the passage of the food and drugs act can not be urged in extenuation of their continuance under the pure-food act. If this were so there would not be a single abuse which the pure-food law was intended to remedy which would not be continued. Granted for the moment, as is shown by the data cited, that the term ice cream before the enactment of the food law and the establishment of the standard did not mean anything. Let it be accorded that it meant any kind of mixture simulating cream which the compounder saw fit to make, provided it was sweet enough and flavored enough to find a purchaser. These facts do not alter the relations of the ice cream to the consumer under the food and drugs act and the standards made in harmony with the act of Congress. It is evident that under that act every name of a food product was intended to represent a certain kind of product and this kind of product is defined and established by the standard. Therefore the protests against the standard as being too high and oppressive to the consumer and impossible of observation by the manufacturer have no basis of fact on which to stand.

A careful study of all the evidence which has been submitted and of the authorities leads to the conclusion that ice cream should be made of cream, that no other ingredient should be used except the sugar and the flavor or fruit, that it should contain not less than 14 per cent of butter fat where concentrated flavors are used and not less than 12 per cent where fruits are used, and with such a definition and standard each consumer will know exactly what he buys and each manufacturer will know exactly what he shall make. If it be desirable to make other frozen puddings, custards, dainties, desserts, etc., at the will of the manufacturer, neither the law nor the standard raises any objections thereto, but these products should be delivered to the consumer under their proper names and not bear the name of a standard product on which the physician and the consumer both rely.

GENERAL CONCLUSIONS.

From a careful study of the data which have been collected it is evident the following conclusions may be drawn:

First. The sanitary conditions of many of the localities where ice cream is manufactured in the District of Columbia are not at all satisfactory. Radical improvements in such localities are necessary to secure purity and freedom from contamination. It is a recognized fact that many cases of violent poisoning which arise from eating cream or ice cream are due to insanitary conditions surrounding the dairy or ice cream factory, the storage for an improper length of time of these products, and the contamination which they suffer by reason of insanitary conditions by infection from preexisting poisonous bodies. The development of ptomaine poisoning in cream and ice cream is entirely prevented by using a fresh sanitary raw product, manufacturing it in perfectly clean surroundings, and disposing of it within a reasonable length of time after manufacture.

Second. The average percentage of fat in the cream sold commercially in the District of Columbia is slightly less than that required by the statute governing the sale of cream in the District of Columbia. It is, however, well within the standard established for cream in general by the Secretary of Agriculture. As long as the Act of Congress relating to the standard of cream in the District of Columbia is on the statute books the dealers should comply with its provisions and cream containing less than 20 per cent of butter fat should not be sold in the District.

Third. The bacteriological examination of cream and ice cream in the District of Columbia shows that much of it contains a number of bacteria which is far in excess of that which should be found in pure uncontaminated fresh materials. This enormous bacterial flora is due to two causes, namely, insanitary conditions of the dairy and factory, and long keeping of the product. From this point of view, therefore, a very large percentage of both cream and ice cream sold in the District of Columbia is highly objectionable.

In regard to its content of butter fat the ice cream sold in the District of Columbia over the period of time mentioned is fairly satisfactory. A very large percentage of all the samples contained more than the 14 per cent of butter fat required for the vanilla type of ice cream and more than 12 per cent of the butter fat required for the fruit type. The establishment of these standards is not subversive to commercial conditions as they existed at the time examinations were made. These standards will, therefore, be regarded not only as reasonable, but as commercially practicable.

Fourth. The use of thickeners in the production of ice cream in the District of Columbia does not appear to be generally practiced. There are many objections to the use of thickeners, the chief of which is that it enables an ice cream to be kept a longer period than it should be. A confection of the character of ice cream is intended for immediate consumption and not for cold storage or long keeping. The ice cream industry is essentially a local industry throughout the country and there is no commercial necessity of transporting ice cream for long distances nor of storing it on board ship, or in other localities for a great length of time. The sooner ice cream can be consumed after it is made the better. Another objection to the thickener is that it aids in the expansion of the volume of cream to proportions entirely beyond the actual amount of nourishment represented; so that, as has been shown in the evidence, from one quart of material two quarts of the product may be produced. Inasmuch as ice cream is sold quite exclusively by volume and not by weight, this expansion can only be regarded as a deception practiced upon the consumer. The use of thickeners of any kind in the manufacture of ice cream is not a commercial necessity. When used the thickener should be wholesome and unobjectionable from a food point of view, and the fact that it has been employed should be plainly stated on the label.

Fifth. The manufacture of frozen dainties containing more or less cream is a legitimate industry, provided all the materials used are pure and wholesome and no false name or appellation is given to the product. A great many products which have been made and sold as ice cream belong to this category. Inasmuch as ice cream is prescribed frequently by physicians for invalids and convalescents, and inasmuch as it is largely eaten by children and others whose stomachs have not full vigor, a definite idea of its composition is necessary to prevent injury and abuse. Hence the term ice cream should be reserved solely for the frozen product consisting of pure, fresh cream, sugar, and a flavor, while appropriate names should be given to other frozen dainties in which more or less cream may enter. The use of milk, skimmed milk, and condensed milk in the manufacture of ice cream does not appear to be advisable or necessary. These substances, when wholesome and pure, are food products of value and their use under appropriate appellations is unobjectionable. Condensed milk diluted to its original volume would not be allowed to be sold as fresh milk under the laws of any of the States or municipalities controlling the milk supply. There seems to be no ethical reason why such products should be permitted to be sold under the name of ice cream. They should be offered to the public under appellations which disclose their real character.

Sixth. The additional regulations which would secure for the District of Columbia a supply of ice cream of unobjectionable quality should look to the restrictions of the materials used to the pure fresh articles. They should require that the butter fat should have a definite percentage corresponding to the established standards of 12 and 14 per cent respectively for the two different types of ice cream. They should protect the consumer against an undue expansion of the ice cream during the process of manufacture so as to make it occupy a volume far larger than is normal. They should restrict the time of storage of ice cream to the limit of ordinary needs of consumption. They should secure absolute cleanliness and neatness in the dairy and in the factory where the ice cream is made. They should exclude from ice cream colors not authorized to be put in foods by the rules and regulations of the food and drugs act. They should exclude from sale ice cream containing a bacterial flora of the enormous proportions exhibited by some of the samples which have been examined. By the adoption of these sanitary regulations an ice cream of standard quality can be offered to the consumers of the District of Columbia, so that anyone purchasing the article may know definitely the character of the material he is buying, the amount which he gets, and may be assured of the freshness and purity of its raw materials and freedom from infection during process of manufacture and the time it is kept in storage.

Seventh. The subject of the pasteurization of milk which is to be used for making ice cream is an important one and should receive careful attention. The data show that cream usually carries a much larger number of organisms than milk. This is probably due chiefly to the fact that the bacteria seem to stick with greater tenacity to the globules of fat than they do to the other parts of the milk. Cream is also often kept longer before being used than milk. The pasteurization, to be effective, should be a thorough one, and the cream pasteurized should be held at the pasteurizing temperature for not less than twenty or twenty-five minutes to insure completion. The ideal cream, of course, is that derived from ideal milk, handled in an ideal manner and used in the shortest possible time in its natural state. Since in the present condition of affairs, however, it is not possible to secure such cream, thorough pasteurization under competent supervision is highly desirable.

TABLE III. –*Chemical, microscopical, and bacteriological examinations of cream from January 30, 1907, to June 12, 1907.*

Date.	Serial No.	Fat.	Fat above (+) or below (−) 18 per cent.	Artificial color.	Bacterial count per cc.	Streptococcus.	Gas production.	Leucocytes.
1907.		*Per ct.*						
Mar. 27	M 4594	18.76	+	....	145,000	No.........	No.........	90,600
May 1	M 4824	22.27	+	....	15,250,000	..do.......	...do....	121,900
May 3	M 4829	23.16	+	...	1,050,000	....do.....	Bubble.....	223,100
Feb. 27	M 4447	16.60		.	57,600	Few......	1 per cent..	1,672,300
Mar. 1	M 4452	18.68	+	...	294,330	...do....	No.........	1,057,200
Mar. 12	M 4511	17.67		..	395,000	...do.....	.....do......	703,300
May 27	M 4952	18.88	+	.	52,500,000	No.........	10 per cent.	486,200
Mar. 1	M 4454	22.00	+	..	1,175,000	Few......	No.........	1,811,500
Apr. 10	M 4680	13.10			1,987,500	.....do....	1 per cent..	386,300
Mar. 2	M 4459	15.07			458,330	Few.......	No.........	21,900
May 27	M 4956	21.38	+		29,500,000	Many..	10 per cent.	22,600
Mar. 7	M 4480	30.00	+	.	2,833,000	...do....	2 per cent...	78,600
Mar. 11	M 4495	21.00	+	.....	862,500	.do....	12 per cent.	33,300
Apr. 2	M 4634	21.82	+		113,000	No.........	3 per cent..	16,000
Apr. 22	M 4766	18.32	+	.	12,500,000	...do....	5 per cent.	57,900
Feb. 20	[a] M 4391	42.43			1,610,000	Few....	....do....	[b]
May 22	M 4932	27.77	+		78,300	No.........	No.........	22,600
Mar. 14	M 4520	19.19	+		161,000	.do....	.do....	46,600
Mar. 25	M 4579	18.77	+		323,750	.do......	...do....	35,300
June 12	B. C. 31	20.55	+		29,500,000	Few	2 per cent..	59,300
May 27	M 4959	16.31	−		6,825,000	No	10 per cent.	60,600
June 11	B. C. 23	24.92	+	.	15,500,000	.do....	2 per cent..	79,300
Feb. 26	M 4434	20.55	+		192,500	.do....	No......	258,900
Mar. 2	M 4462	20.82	+	..	271,660	Few... .	5 per cent..	228,400
Mar. 8	M 4484	19.13	+		200,000	..do..	No.. ..	77,200
Mar. 23	M 4565	18.56	+	.	8,375,000	.do....	.....do...	57,300
Jan. 30	M 4328	12.60	−	Colored.	171,830	......	Not det...	337,500
Feb. 1	M 4330	12.90		. do ...	938,300	Few... .	..do....	465,400
Feb. 4	M 4339	12.60		.do. ..	354,800	.do....	...do....	403,000
Mar. 20	M 4548	22.31	+		136,000	.....do....	No...	978,300
June 11	B. C. 19	21.66	+		155,000,000	Many..	10 per cent	1,226,000
Mar. 6	M 4471	22.75	+		245,830	Few....	..do..	113,200
Do..	M 4472	21.95	+		288,300	.....do....	No..	111,900
Apr. 19	M 4747	15.42	−		99,000	No..	Bubble .	43,300
May 25	M 4951	20.08	+		25,600	do....	15 per cent	56,600
Feb. 18	M 4381	17.58			8,050,000	.do.. .	No..	159,100
Feb. 20	M 4387	16.81			296,660	Few...	.do. .	318,300

[a] Double cream excluded from average.

[b] Cloudy, impossible to count.

Table III.—*Chemical, microscopical, and bacteriological examinations of cream from January 30, 1907, to June 12, 1907*—Continued.

Date.	Serial No.	Fat.	Fat above (+) or below (−) 18 per cent.	Artificial color.	Bacterial count per cc.	Streptococcus.	Gas production.	Leucocytes.
1907.		*Per ct.*						
Apr. 27	M 4804	16.95	−	....	753,300	Free.......	No	126,500
June 12	B. C. 33	18.19	+	....	....	....	....	127,900
Feb. 18	M 4383	19.45	+	....	46,166,000	No.........	No......	654,000
Feb. 20	M 4393	20.42	+	....	4,400,000	Many.......	...do....	113,900
Mar. 30	M 4616	19.17	+	....	4,825,000	...do.....	...do.....	3,565,300
May 1	M 4822	17.20		....	320,800	Few........	...do.....	968,400
May 29	M 4979	17.58	−	....	9,250,000	No.....	...do....	2,186,600
Apr. 13	M 4716	16.63		....	9,750,000	.do....	27 per cent.	1,837,300
Apr. 24	M 4780	18.33	+	.....	425,000	Few......	25 per cent.	4,082,600
May 24	M 4930	17.43	−	....	575,000	No...	4 per cent...	2,534,600
Mar. 20	M 4540	19.94	+	....	28,900,000	Many.	No.........	130,500
Apr. 23	M 4777	41.75	+	.....	1,375,000	No..........	1 per cent..	70,600
Mar. 1	M 4448	18.25	+	...	2,066,600	Many....	17 per cent.	156,100
Mar. 23	M 4567	19.06	+	.	70,400,000	Few.......	No.........	121,200
Mar. 26	M 4581	18.02	+	....	161,000	Many.....	Bubble....	309,000
Apr. 6	M 4666	20.50	+	....	2,087,500	....do.....	40 per cent.	219,100
Apr. 29	M 4813	23.80	+	.	30,500,000	Few.......	2 per cent..	57,300
Mar. 11	M 4500	14.04	−	Colored.	180,000	No..........	No.........	1,262,000
Apr. 26	M 4799	24.16	+	....	309,000,000	Few.......	3 per cent..	[a]
Mar. 18	M 4531	20.96	+	.	516,000	No..........	No.........	41,300
Mar. 22	M 4554	18.97	+	...	3,350,000	...do....	.do....	44,600
Apr. 15	M 4721	23.17	+	.....	29,000,000	.do.....	...do....	6,000
June 12	B. C. 34	20.06	+	....	1,177,500	Few......	.do....	29,300
Mar. 11	M 4498	18.02	+	....	359,960	....do.	Bubble.....	57,300
Mar. 12	M 4502	17.95	−	....	350,830	No..........	No.........	23,900
Mar. 13	M 4512	18.21	+	....	397,500	Many.	.do....	75,900
Mar. 22	M 4559	19.16	+	......	[b]	No......	.do....	57,900
Mar. 15	M 4528	19.18	+	......	90,000	Few......	.do....	39,300
Mar. 20	M 4543	20.92	+	....	12,000	No..........	.do....	35,300
Apr. 1	M 4628	21.50	+	......	593,000	Few....	10 per cent	1,374,000
Apr. 27	M 4806	14.91	−	.....	59,500,000	No..........	Bubble.....	1,356,600
May 3	M 4827	23.56	+	...	700,000	.do....	No.........	1,094,600
Mar. 27	M 4593	15.90	−	...	5,400,000	Many...	..do....	36,600
Mar. 19	M 4536	16.18	−	....	54,000,000	Few........	.....do.....	73,200
May 25	M 4949	22.33	+	....	151,600	No........	25 per cent.	22,600
Mar. 15	M 4524	16.95	−	.	1,450,000	.do.	No.........	32,600
Mar. 19	M 4533	16.84		.	1,520,800	Many.......	.....do.....	217,100
Mar. 25	M 4575	17.46	−	.	755,860	Few........	Bubble....	145,800
Feb. 26	M 4429	17.00	−	..	180,000	No..	No........	148,600
Feb. 27	M 4446	15.97	−	.	208,000	Very many.	12 per cent.	58,900
Apr. 20	M 4758	15.84	−		61,000	No........	10 per cent	163,800
May 24	M 4928	18.08	+		625,000	.....do......	6 per cent..	180,500
Feb. 13	M 4366	15.25	−		21,216,000	Numerous..	18 per cent.	361,800
Mar. 3	M 4487	17.00			275,000	Many.......	3 per cent..	561,400
Apr. 2	M 4629	17.19	−		537,500	No..	4 per cent	2,237,300
Apr. 22	M 4768	20.02	+		17,500,000	.do...	3 per cent..	321,000
May 29	M 4976	18.38	+		26,000,000	.do	25 per cent	583,400
Feb. 23	M 4426	18.02	+		350,830	.do	15 per cent	2,446,000
Mar. 2	M 4464	23.70	+		1,183,000	Few	8 per cent.	1,796,800

[a] Too badly coagulated to count.

[b] Not determined.

TABLE III.—*Chemical, microscopical, and bacteriological examinations of cream from January 30, 1907, to June 12, 1907*—Continued.

Date.	Serial No.	Fat.	Fat above (+) or below (—) 18 per cent.	Artificial color.	Bacterial count per cc.	Streptococcus.	Gas production.	Leucocytes.
1907.		*Per ct.*						
Feb. 15	M 4372	15.40			1,408,330	Many	12 per cent	476,400
Feb. 20	M 4388	17.29	–		159,330	Few	No	2,219,100
Mar. 13	M 4514	17.91	–		175,000	do	do	571,400
May 27	M 4958	13.62	–		20,500,000	No	40 per cent	383,600
Feb. 15	M 4371	15.25	–		1,888,330	Many	4 per cent	118,200
Mar. 12	M 4509	20.10	+		200,000	No	No	122,500
Mar. 19	M 4538	18.00	+		6,675,000	Many	do	132,500
May 18	M 4907	18.10	+		40,500,000	No	3 per cent	91,200
May 25	M 4948				18,000,000	Few	20 per cent	220,400
Feb. 6	M 4345	18.80	+		262,000	No	12 per cent	23,600
Do.	M 4346	18.90	+		631,660	Few	2 per cent	25,800
Feb. 8	M 4350	17.30	–		260,000	do	No	1,556,300
Feb. 9	M 4360	17.10	–		222,660	Very few	do	760,600
Feb. 20	M 4392	18.00	+		150,160	No	do	534,800
Apr. 24	M 4784	26.42	+		2,825,000	do	do	825,800
May 24	M 4926	30.52	+		1,300,000	do	8 per cent	2,348,000
Feb. 20	M 4389	16.34			16,966,000	Many	No	535,400
Mar. 1	M 4451	14.66	–		43,600,000	do	17 per cent	297,300
Mar. 15	M 4525	21.30	+		87,000	No	No	18,000
June 12	B. C. 32	23.70	+					28,000
Feb. 26	M 4428	13.38	–		2,123,300	Very many	2 per cent	407,300
Apr. 20	M 4760	16.74			50,500,000	Few	No	104,600
May 28	M 4965	15.65	–		11,150,000	No	45 per cent	29,300
Feb. 4	M 4337	19.35	+	Colored	560,330	Many	Not det.	107,000
Feb. 5	M 4342	18.90	+	do	930,000	Numerous	12 per cent	148,200
Feb. 6	M 4348	19.33	+	do	9,116,000	Few	40 per cent	83,300
Mar. 5	M 4465	18.45	+	do	2,041,600	Many	15 per cent	197,800
Mar. 6	M 4469	18.50	+	do	2,841,600	do	10 per cent	194,500
Mar. 9	M 4492	18.58	+	do	1,070,830	Few	No	113,200
Mar. 13	M 4517	20.60	+	do	543,300	Many	do	100,500
Apr. 5	M 4643	16.35	–		4,300,000	No	do	1,552,000
May 27	M 4954	14.56	–		26,500,000	Few	28 per cent	500,200
Feb. 16	M 4377	15.40		Colored	262,330	No	No	335,000
Feb. 18	M 4382	14.10	–	do	202,350	do	do	59,300
Feb. 20	M 4394	14.77		do	269,330	do	do	168,500
Apr. 15	M 4723	22.69	+	do	126,600	do	4 per cent	1,854,000
May 27	M 4957	20.20	+		11,500,000	do	12 per cent	12,700
Feb. 20	M 4390	18.48	+		14,933,000	Very few	3 per cent	134,500
Apr. 1	M 4624	19.59	+		14,000,000	Many	No	526,100
Feb. 26	M 4430	20.20	+		16,966,000	do	4 per cent	103,900
Apr. 8	M 4673	15.86	–		7,250,000	Few	5 per cent	107,900
Apr. 16	M 4729	20.33	+		725,000	No	3 per cent	97,900
May 28	M 4964	25.06	+		15,500,000	do	30 per cent	77,900
Mar. 27	M 4596	25.98	+		5,800,000	Few	5 per cent	690,600
Apr. 3	M 4639	15.91			27,000	No	No	36,600
May 1	M 4819	22.60	+		61,500,000	do	15 per cent	445,600
May 29	M 4977	18.12	+		64,000,000	Few	20 per cent	238,400

TABLE IV.—*Chemical, microscopical, and bacteriological examinations of ice creams from April 1 to August 1, 1907.*

Date.	Serial No.	Variety.	Fat.	Foreign additions.	Bacteria per cc.	Streptococcus.	Gas production.	Leucocytes.	Fat above (+) or below (−) standard.
1907.			*Per cent.*						
Apr. 26	M 4786	Vanilla	13.77	Vegetable jelly	8,850,000	No	No		−
June 4	M 4992	do	21.43		712,500	do	4 per cent		+
May 13	M 4869	do	3.37		3,675,000	do	No		−
May 14	M 4876	do	3.46		5,500,000	do	3 per cent		−
May 16	M 4887	do	16.19		40,500,000	do	do		+
May 27	M 4961	do	16.15		110,000,000	do	20 per cent		+
May 29	M 4981	Chocolate	14.41		43,750,000	do	30 per cent		+
Do	M 4982	Strawberry	10.71		15,375,000	do	12 per cent		−
Do	M 4983	Vanilla	14.37		30,000,000		No		+
	B C 5				17,750,000		7 per cent		
	B C 6				28,750,000		15 per cent		
	B C 7				4,250,000		3 per cent		
July 17	B C 220	Strawberry	13.46		6,250,000		40 per cent		+
Do	B C 221	Peach	11.93		25,625,000		30 per cent		−
Do	B C 222	Vanilla	12.08		17,625,000	Few	35 per cent		−
Do	B C 223	Chocolate	13.77		21,250,000		28 per cent		+
June 14	B C 44	do	18.36		960,000		No		+
July 10	B C 162	Vanilla	19.99		1,101,000		do		+
Do	B C 163	Chocolate	19.86		5,800,000		30 per cent		+
Do	B C 164	Strawberry	11.26		8,500,000		35 per cent		−
Apr. 26	M 4748	Strawberry	11.59	Gelatin	6,330,000		No		−
Do	M 4749	Vanilla	12.87	do	2,800,000	Few	do		−
Do	M 4750	Chocolate	13.42	do	2,083,000		1 per cent		+
May 8	M 4844	Vanilla	15.30		7,000,000		No		+
Apr. 6	M 4654	do	15.96		364,000,000	Many	2 per cent	83,300	+
Do	M 4655	Strawberry	12.71		26,500,000	No	No		+
	M 4656				75,000,000	do	do		

TABLE IV.—*Chemical, microscopical, and bacteriological examinations of ice creams from April 1 to August 1, 1907*—Continued.

Date.	Serial No.	Variety.	Fat	Foreign additions.	Bacteria per cc.	Streptococcus.	Gas production.	Leucocytes.	Fat above (+) or below (—) standard.
1907			*Per cent.*						
May 27	M 4603	Vanilla	15.10		155,000,000		38 per cent		+
June 14	B. C. 42	do	13.91		733,000	Few	No		—
	M 4604	Strawberry	10.73		8,675,000	No	do		—
	M 4605				714,000	Few	do		
	M 4606	Vanilla	19.47		7,250,000	do	do		+
	M 4607	Strawberry	8.59		5,033,000	do	do		—
	M 4608	Chocolate	10.74		6,375,000	do	do		—
	M 4649	Vanilla	13.82		1,078,300	do	do		—
Apr. 25	M 4771	Tru-cream	11.33		276,000	No	do		—
	M 4772				1,300,000	do	do		
May 7	M 4845	Vanilla	13.75	Vegetable jelly	7,800,000	do	do		—
May 9	M 4888	do	14.33		3,250,000		20 per cent		+
	B. C. 11				1,016,000		60 per cent		
July 23	B. C. 233	Vanilla	7.77	Starch and gelatin	3,250,000		28 per cent		—
May 24	M 4919	do	12.74		19,750,000	Few	No		—
June 15	B. C. 50	do	14.12		2,340,000	No	6 per cent		+
June 21	B. C. 74	do	15.27		3,850,000	Few	15 per cent		+
	M 4584				51,125,000	Many	3 per cent		
	M 4658	Strawberry			1,441,600	No	No		
Apr. 8	M 4659	Vanilla	15.00		1,466,000	do	do		+
Do	M 4660	Chocolate	18.27		29,625,000	do	3 per cent		+
July 2	B. C. 139	Strawberry	13.14		11,000,000	do	4 per cent		+
July 24	B. C. 234	Vanilla	13.61		1,682,000	do	7 per cent		—
Do	B. C. 235	Chocolate	21.20		3,975,000		4 per cent		+
Do	B. C. 236	Strawberry	14.48		1,780,000		14 per cent		+
Do	B. C. 237	Peach	13.58		3,475,000		do		+
Apr. 20	M 4751	Chocolate	7.82	Vegetable jelly	19,500,000		15 per cent		—

Apr. 22	M 4752	Vanilla	6.71	do.	3,300,000	Many	76 per cent		−
May 8	M 4843	[illegible]	6.81	Starch and vegetable jelly	2,370,000		5 per cent		−
July 31	B. C. 165	[illegible]	6.99	do.	2,883,000	Few	45 per cent		−
Do.	B. C. 166	Chocolate	7.00	do.	1,175,000		20 per cent		−
June 27	B. C. 48	Vanilla	14.93		34,750,000	Many	No		+
June 21	B. C. 7	do.	12.83		5,650,000	No	do.		−
May 10	M 4867	do.	16.75		8,160,000	do.	3 per cent		+
May [illegible]	M 4880	do.	16.05		31,000,000	do.	2 per cent		+
May [illegible]	M 4902	do.	15.79		85,000,000	do.	12 per cent		+
May [illegible]	M 4917	do.	8.63		4,235,000	Few	No		−
Do.	M 4948	Strawberry	5.61		8,750,000		½ per cent		−
June 28	B. C. 122	do.	7.17		14,700,000		1 per cent		−
Apr. [illegible]	M 4856	Vanilla	16.01	Gelatin	10,750,000	Few	3 per cent	65,000	+
Do.	M 4858	Strawberry	13.56	do.	33,460,000		6 per cent		+
Do.	M 4857	Chocolate	14.83		31,000,000		4 per cent		+
May [illegible]	M 4852	Vanilla	13.62		10,750,000		1 per cent		+
June 27	B. C. 112	Strawberry	9.89		26,875,000		12 per cent		−
[illegible]	B. C. 224	Vanilla	11.02	Gelatin	8,666,600		40 per cent		−
Mar. [illegible]	M 4546				59,125,000	Many	No	80,600	
Do.	M 4750				61,625,000	do.	do.	62,600	
Apr. [illegible]	M 4622	Vanilla	11.49		14,600,000	Few	do.	33,300	−
Apr. 25	M 4787	do.	13.90	Vegetable jelly	50,000,000	No	do.		−
[illegible]	B. C. 4	do.		Gelatin	42,500,000	Few	1 per cent		
May 17	M 4890	Chocolate	13.09		34,500,000		3 per cent		+
Do.	M 4894	Vanilla	13.68		19,000,000		No		−
May [illegible]	M 4865	Strawberry	11.78	Coal-tar color	8,500,000		3 per cent		−
June 4	M 4902	Vanilla	14.67		25,750,000	No	25 per cent		+
June 4	B. C. 45	do.	2.82		1,516,000	Few	5 per cent		−
[illegible]	B. C. 88	do.	8.83		9,750,000	Many	50 per cent		−
Do.	B. C. 180	Chocolate	2.60		18,000,000		47 per cent		−
May [illegible]	M 4907	Vanilla	14.79		31,000,000		6 per cent		+
[illegible]	B. C. 228	do.	10.94	Gelatin	10,750,000	No	15 per cent		−
Do.	B. C. 226	Peach	9.87	do.	15,500,000	do.	do.		−
Apr. [illegible]	M [illegible]	do.	14.14		335,000,000		1 per cent		+
Do.	M [illegible]	Vanilla	14.65		26,500,000		4 per cent		+

TABLE IV.—*Chemical, microscopical, and bacteriological examinations of ice creams from April 1 to August 1, 1907*—Continued.

Date	Serial No.	Variety.	Fat	Foreign additions	Bacteria per cc.	Strepto-coccus.	Gas production.	Leucocytes.	Fat above (+) or below (−) standard.
1907.			*Per cent*						
Apr. 19	M 4740	Chocolate	14.77		65,000,000		10 per cent		+
Do	M 4741	Strawberry	11.92		37,000,000		No		−
Apr. 26	M 4789	Vanilla	7.21	Vegetable jelly	49,000,000		do		−
June 27	B. C. 116	Strawberry	12.71	Gelatin	23,916,600		30 per cent		+
	M 4585				27,250,000	Few	6 per cent		
	M 4586				5,625,000	do	No		
June 14	B. C. 43	Vanilla	6.29		2,825,000	do	do		−
July 12	B. C. 190	do	12.03		17,750,000		65 per cent		−
June 17	B. C. 54	do	16.90		32,500,000	Many	No		+
	M 4997				21,500,000		do		
	M 4998				28,750,000		2 per cent		
	M 4999				18,250,000		25 per cent		
Apr. 23	M 4761	Vanilla	13.97		1,620,000	No	Bubble		−
Do	M 4762	Strawberry	10.28		6,500,000	do	12 per cent		−
Do	M 4763	Chocolate	15.99		633,000	do	2 per cent		+
June 4	M 4963	Strawberry	15.12		6,783,700		5 per cent		+
Do	M 4964	Vanilla	15.96		1,210,000		1 per cent		+
June 27	B. C. 113	Strawberry	10.65		9,133,300		25 per cent		−
Apr. 16	M 4733	Vanilla	8.83		10,750,000		2 per cent		−
May 16	M 4884	do	10.62	Vegetable jelly	7,016,000		No		−
June 24	B. C. 94	do	12.22	do	30,875,000	No	12 per cent		−
Do	B. C. 95	Strawberry	10.95	do	22,500,000		20 per cent		−
Do	B. C. 96	Chocolate	12.78	do	32,750,000		12 per cent		+
July 17	B. C. 219	Vanilla	14.90	do	3,450,000	No	25 per cent		+
May 23	M 4913	Strawberry	13.42		22,500,000		2 per cent		+
Do	M 4914	Vanilla	16.56		18,500,000	No	1 per cent		+
Do	M 4915	Chocolate	18.28		48,750,000		2 per cent		+

	B. C. 8				20,000,000		7 per cent	
	B. C. 9				23,500,000	No	No	
	B. C. 10				24,500,000	do	12 per cent	
June 22	B. C. 82	Chocolate	14.84		6,000,000		7 per cent	+
June 24	B. C. 83	Strawberry	10.53		5,250,000		No	−
Do	B. C. 84	Vanilla	13.72		4,300,000	Few	do	−
June 25	B. C. 100	do	14.73		169,750,000		30 per cent	+
June 14	B. C. 47	do	19.29		26,250,000	Few	No	+
July 12	B. C. 187	do	12.56		5,000,000	No	40 per cent	−
July 15	B. C. 201	do	15.51		8,250,000		2 per cent	+
Apr. 22	M 4753	Chocolate	13.93	Starch	43,000,000		1½ per cent	+
Do	M 4754	Vanilla	13.72	do	44,250,000		8 per cent	−
May 9	M 4851	do	14.12	Starch and vegetable jelly	38,250,000		No	+
June [illegible]	M 4996	Chocolate	13.37	Gelatin	16,750,000		5 per cent	+
Do	M 4997	Strawberry	12.23	do	7,000,000		10 per cent	+
Do	M 4998	Vanilla	12.82	do	12,250,000	Many	4 per cent	−
Apr. 16	M 4731	do	20.78	do	6,566,000	Few	2 per cent	+
May 15	M 4880	do	17.67	do	10,750,000	No	30 per cent	+
	M 4735				11,833,300		No	
Apr. 18	M 4736	Vanilla	14.42		13,125,000		Bubble	+
Do	M 4737	Chocolate	12.17		14,750,000		3 per cent	+
Apr. 25	M 4788	Vanilla	13.16		7,500,000		55 per cent	−
June 27	B. C. 117	Strawberry	8.75		4,191,600		20 per cent	−
May 28	M 4897	Special vanilla	15.12		12,500,000	No	6 per cent	+
Do	M 4898	Soda fountain vanilla	11.12		11,000,000	do	No	−
July 15	B. C. 202	Vanilla	12.14	Starch and vegetable jelly	55,250,000	do	15 per cent	−
June 25	B. C. 98	do	9.70		7,616,600	Many	5 per cent	−
June [illegible]	B. C. [illegible]	do	16.54		6,000,000	No	No	+
July 19	B. C. 225	do	7.47	Gelatin	16,750,000		25 per cent	−
Apr. [illegible]	M 4732	Frozen dainties	6.53	Starch and vegetable jelly	17,000,000		4 per cent	−
	M 4790			do	14,500,000		2 per cent	
Apr. [illegible]	M 4791	Frozen dainties	4.91	do	137,500		1 per cent	−
	M 4792			do	162,500		No	
[illegible] 28	B. C. 125	Strawberry	2.80	Starch, gelatin, and vegetable jelly	11,900,000		30 per cent	−
[illegible] 7	B. C. 52	Vanilla	13.90	Gelatin	68,000,000	No	6 per cent	−

TABLE IV.—*Chemical, microscopical, and bacteriological examinations of ice creams from April 1 to August 1, 1907*—Continued.

Date.	Serial No.	Variety.	Fat.	Foreign additions.	Bacteria per cc.	Strepto-coccus.	Gas production.	Leucocytes.	Fat above (+) or below (−) standard.
1907.			*Per cent.*						
[illegible]	B. C. 42	Do.	17.45		4,250,000	Many	1 per cent.		+
[illegible] 24	B. C. 258	Hokey-pokey	2.84		2,513,300		60 per cent	...	−
[illegible] 22	B. C. 80	Vanilla	14.56		1,060,000		No.		+
Do.	B. C. 81	Strawberry	13.29		12,750,000		50 per cent		+
[illegible]	B. C. 225	Vanilla	17.95		7,766,600	No.	40 per cent		+
Do.	B. C. 226	Strawberry	16.84		976,000		45 per cent		+
Do.	B. C. 227	Peach	14.34		7,633,300		35 per cent		+
	B. C. 25[illegible]				365,000,000		3 per cent		
[illegible] 29	B. C. 27[illegible]	Vanilla	17.31		53,500,000		10 per cent		+
[illegible] 4	M. 4[illegible]	Do.	12.16	Gelatin.	26,266,600	No.	3 per cent.		−
	B. C. 12			Do.					
[illegible] 27	B. C. 247	Vanilla	14.09	Starch and vegetable jelly	295,000,000		10 per cent		+
Do.	B. C. 248	Chocolate	14.92	Do.	325,000,000		15 per cent.		−
Do.	B. C. 249	Strawberry	11.62	Do.	270,000,000		37 per cent		
July 29	B. C. 251	Chocolate	14.78	Do.	51,500,000		8 per cent.		+
Do.	B. C. 252	Strawberry	10.70	Do.	20,800,000		4 per cent		−
May [illegible]	M. 485	Velvet kind C. A. S. vanilla	13.87		983,000		1 per cent		−
[illegible] 24	B. C. 226	Vanilla	10.39	Gelatin.	9,375,000	No.	30 per cent		−
Do.	B. C. 227	Strawberry	8.54	Do.	7,091,600		60 per cent		
[illegible] 27	B. C. 240	Vanilla	13.33		3,450,000		No.		
Do.	B. C. 2[illegible]	Chocolate	14.69		3,680,000		Do.		+
Do.	B. C. 241	Strawberry	12.17	Coal tar dye	950,000		95 per cent		+
Do.	B. C. 242	Coffee	14.94		1,850,000		40 per cent		+
June 15	B. C. 49	Vanilla	16.83	Vegetable jelly	33,250,000		45 per cent		−
	B. C. 75			Do.[a]	8,000,000	No.	25 per cent		

[a] Cooked before freezing.

June 14	B. C. [illegible]	Vanilla	13.70		5,500,000	Few	No	−
June 18	B. C. 56	do	12.02		6,500,000	No	15 per cent	−
	M 4773				1,866,000	do	6 per cent	−
May [illegible]	M 4883	Vanilla	9.81		16,500,000	Many	1 per cent	
July [illegible]	B. C. 218	do	11.10		204,000,000	No	25 per cent	−
June 13	B. C. 37	do	11.72	Vegetable jelly	8,600,000	do	No	−
July 13	B. C. 197	do	15.67		115,000,000	do	45 per cent	+
Do.	B. C. 198	Strawberry	15.42		165,000,000		50 per cent	+
	M 4589				18,500,000	No	No	
	M 4590				7,825,000	do	do	
	M 4591				2,625,000	do	1 per cent	
July 15	B. C. 199	Strawberry	12.69		8,500,000		18 per cent	+
Do.	B. C. 200	Chocolate	14.60		25,250,000		10 per cent	+
May 20	M 4858	Vanilla	13.99		391,600		No	+
Do.	M 4859	Chocolate	13.77		1,150,000		do	+
Do.	M 4860	Strawberry	8.68		1,307,500		do	−
May 28	M 4968	do	14.94		105,000,000		10 per cent	+
Do.	M 4969	Vanilla	14.93		115,000,000	No	15 per cent	+
Do.	M 4970	Chocolate	15.34		95,000,000		2 per cent	+
June 29	B. C. 129	Strawberry	12.77		11,950,000		No	+
July 1	B. C. 154	Vanilla	17.28		12,416,600		10 per cent	+
May 18	M 4899	do	15.28		17,250,000	Few	5 per cent	+
	M 4900				16,500,000		½ per cent	
May 28	M 4901	Chocolate	14.96		23,750,000		2 per cent	+
Apr. 11	M 4682	Vanilla	12.45	Starch	10,500,000	Few	No	−
Apr. 12	M 4683	Chocolate	12.16	do	15,500,000		7 per cent	+
Do.	M 4684	Strawberry	12.30	do	11,500,000		60 per cent	+
May 24	M 4951	Vanilla	17.34	do	31,500,000	Few	15 per cent	+
	B. C. 111				11,235,000		25 per cent	
Apr. 4	M 4645	Strawberry	4.82	Gelatin	42,000,000	Few	1 per cent	−
Apr. 5	M 4646	Chocolate	6.74	do	43,500,000		3 per cent	−
May 15	M 4881	Vanilla	8.25	do	9,000,000		20 per cent	−
Do.	M 4882	Chocolate	8.47	do	6,500,000		3 per cent	−
May 14	M 4868	Hokey-Pokey	4.86		73,250,000		No	−
May 29	M 4904	Vanilla	15.26	Starch and vegetable jelly	960,000	No	3 per cent	+

TABLE IV.—*Chemical, microscopical, and bacteriological examinations of ice creams from April 1 to August 1, 1907*—Continued.

Date.	Serial No.	Variety.	Fat.	Foreign additions.	Bacteria per cc.	Strepto-coccus.	Gas production.	Leucocytes.	Fat above (+) or below (−) standard.
1907.			*Per cent*						
May 4	M 4853	Chocolate[a]	12.26		1,650,000		No		+
Do	M 4854	Strawberry[a]	12.72		626,000		1 per cent.		+
Do	M 4855	Vanilla[a]	15.54		560,000		No		+
May 10	M 4857	do[a]	14.08		[illegible]		50 per cent.		+
June 29	B. C. 128	Strawberry[a]	9.06	Starch and vegetable jelly	5,841,600		15 per cent		
July 2	B. C. 135	Vanilla[a]	11.79	do	4,475,000		No		−
June 13	B. C. 38	Vanilla	14.16		13,000,000	Few	do		+
July 22	B. C. 230	do	13.63		37,500,000	No	20 per cent		−
Do	B. C. 231	Chocolate	12.47		30,000,000		18 per cent		+
July 29	B. C. 243	Vanilla	13.61		23,000,000		1 per cent.		−
Do	B. C. 244	Strawberry	8.82		3,330,000		do		
Do	B. C. 245	Peach	11.03		7,500,000		3 per cent.		−
May 16	M 4889	Vanilla	10.15		3,300,000		1 per cent.		−
May 20	M 4902	do	16.07		11,750,000	No	No		+
Do	M 4903	Vanilla, milk ice cream	4.13		820,000	do	4 per cent.		−
May 25	M 4946	Vanilla	13.18		1,375,000	do	No		−
June 3	M 4988	do	11.54		2,525,000		20 per cent		−
May 17	M 4890	Strawberry	15.93	Gelatin	59,250,000		1 per cent		+
Do	M 4891	Vanilla	15.67	do	2,900,000	No	No		+
Do	M 4892	Chocolate	14.23	do	3,575,000		5 per cent.		+
June 4	M 4900	Vanilla	16.60	do	2,005,000	Many	No		
July 2	B. C. 137	Strawberry	14.61	do	5,308,300	No	20 per cent.		+
Apr. 9	M 4967	Vanilla	13.64		1,858,300		No		
	M 4968				37,000,000		do		
Apr. 9	M 4969	Strawberry	13.09		1,433,000		do		+

[a] C. & S. ice cream.

May 15	M 4878	Chocolate	17.11		850,000		do.	+
Do.	M 4879	Vanilla	15.28		1,083,000		do.	+
July 2	B. C. 138	Strawberry	14.44		26,750,000		20 per cent.	+
Apr. 25	M 4785	Vanilla	12.67		7,000,000		2 per cent.	—
May 18	M 4896	do.	10.51		32,500,000	Many	do.	—
June 13	B. C. 39	do.	14.36		65,000,000	No	No	+
Apr. 19	M 4742	do.	4.58		9,750,000	do.	Bubble	—
May 14	M 4877	do.	5.06		11,000,000	do.	12 per cent.	—
May 25	M 4916	do.	8.09	Starch and vegetable jelly.	195,000,000	Many.	2 per cent.	
May 7	M 4842			do.	390,250		No.	
June 25	B. C. 99	Vanilla	11.89		10,330,300	No.	18 per cent	—
June 13	B. C. 40	do.	6.92	Starch and vegetable jelly	12,000,000	do.	6 per cent.	—
July 13	B. C. 195	do.	7.15	do.	2,016,000	Few	20 per cent	—
Do.	B. C. 196	Chocolate.	7.38	do.	2,550,000		45 per cent	—
May 9	M 4848	Vanilla	13.40	do.	23,750,000		No	—
Do.	M 4849	Strawberry	12.73	do.	9,375,000		1 per cent.	+
Do.	M 4850	Chocolate	15.73		1,716,000		½ per cent.	+
June 28	B. C. 121	Strawberry	15.90		22,500,000		50 per cent	+
June 29	B. C. 127	Vanilla.	13.66	Starch and vegetable jelly	131,750,000		25 per cent	—
July 3	B. C. 167	do.	10.90	Gelatin	12,000,000	Few	40 per cent	—
Do.	B. C. 168	Chocolate.	11.47	do.	25,000,000		35 per cent.	—
July 23	B. C. 232	Vanilla	9.86		29,500,000		30 per cent	—
May 29	M 4905	do.	8.85	Starch and vegetable jelly	3,250,000	No.	1 per cent	—
June 6	M 4909	Strawberry	15.46		21,750,000		2 per cent.	+
Do.	B. C. 1	Vanilla.	14.93		14,500,000	Few	6 per cent.	+
	B. C. 2				5,125,000		3 per cent.	
	B. C. 3	Chocolate			11,500,000		1 per cent.	
June 15	B. C. 51	Vanilla	15.69		2,450,000		do	+
	M 4587				380,000	No.	No.	

10. THE CHEMISTRY OF MILK.

THE CHEMISTRY OF MILK.

By Joseph H. Kastle, *Chief Division of Chemistry;* and Norman Roberts, *Passed Assistant Surgeon, Public Health and Marine-Hospital Service.*

PREFACE.

In the following pages the attempt has been made to present succinctly and yet sufficiently comprehensively for a thorough understanding of the subject what is known to-day regarding the chemistry of milk, and to give the results of the analyses of the Washington milk supply, made in the division of chemistry of the hygienic laboratory during a period of twelve weeks extending through July, August, and September, 1907.

Part I of this communication deals with the chemical composition and general characteristics of milk.

Part II deals with the chemical changes occurring in milk. Under this head are included changes in the composition of milk brought about, (1) by the action of heat and acids, (2) by the action of the enzymes of milk, (3) by the action of the digestive ferments, (4) by the action of bacteria and other micro-organisms, including the lactic acid fermentation and the abnormal fermentations of milk. Under this last section a few pages are also devoted to the subject of poisoning by milk, galactotoxismus. Under section 1 the destructive effect of heat on the milk enzymes is also considered.

Part III is devoted to the consideration of legal standards governing the sale of milk in various localities.

Part IV is devoted to the subject of milk adulteration, by skimming, watering, and the addition of foreign substances, including artificial coloring matters and milk preservatives. Some attention has been paid to the effect of artificial coloring matters and preservatives on the health of man.

Part V is devoted to the general consideration of the Washington milk supply. Under this head will be found, (1) a brief outline of the methods employed in milk analysis, (2) the results of our analyses of the Washington milk supply, (3) conclusions regarding the general character of the Washington milk supply.

In the preparation of this communication we have drawn freely from the writings of numerous authors on the subjects herein considered. In every instance the attempt has been made to give due credit to all concerned and no special credit is claimed for any originality in the treatment of any of the subjects herein presented. Free use has been made of many treatises and works on the subject of milk and milk analysis and of many original articles and monographs treating of the composition of milk, the rennin coagulation, the milk ferments, the use of coloring matters and preservatives and their possible injurious effects. For all of these due acknowledgment is hereby made. To Conn, "Agricultural Bacteriology," Philadelphia, 1901, we are especially indebted for much on the subject of the abnormal fermentations of milk. To Leach, "Food Inspection and Analysis," New York, 1907, and to Van Slyke, "Modern Methods of Testing Milk and Milk Products," New York and London, 1907, for methods pertaining to milk analysis, and for valuable data on the composition of milk and milk adulteration. To the health office of the District of Columbia we are indebted for much assistance during the progress of the work, and to Prof. Victor C. Vaughan, of the University of Michigan, for private information relative to recent progress in the field of milk poisons.

PART I.—THE COMPOSITION AND GENERAL CHARACTERISTICS OF MILK.

Milk is the specific secretion of the mammary glands.[a] The milk of a number of animals has been and is still very extensively used as food by man. The milk of different animals shows a general agreement in physical properties and composition, containing essentially the same ingredients but exhibiting differences in the amounts of the several constituents. Of all the different kinds of milk, that of the cow is the most universally used, and in what follows, unless expressly stated to the contrary, it will be understood that cow's milk is meant whenever the term "milk" is employed.

In the perfectly fresh state, milk is a yellowish-white, opaque fluid. When allowed to stand undisturbed for some time it separates into two distinct layers. The upper, lighter layer, occupying a smaller volume than the lower, heavier layer, is what is called "cream," and consists largely of globules of fat. The lower, heavier layer, white or bluish white in color, is when separated known as "skim milk." On account of changes due to the growth and action of micro-organisms the color of the milk may be altered; for example, it has been found under certain conditions to become red, blue, yellow, etc. As is well known, milk when fresh possesses a distinctly sweet taste and a characteristic odor. It is heavier than water, the specific gravity of cow's milk ranging from 1.027 to 1.035. It freezes at a temperature somewhat lower than the freezing point of water—according to Beckmann (1), at −0.554° C. Atkins (2) has also found the freezing point of milk to be practically constant, viz. −0.55° C., the variations from this mean value rarely exceeding 0.03° C.

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[a] Ordinarily milk is secreted by the female mammal only, and only after parturition. In some instances, however, the mammae of newborn children, males as well as females, also secrete small amounts of a milk-like fluid known as witch's milk; and still more rarely milk is said to have been secreted by the mammary glands of the adult human male. Fluids resembling milk are also formed in certain pathological conditions. All of these instances are, however, more or less rare and warrant no further consideration in this connection. Milk-like secretions of vegetable origin are also not considered in this communication.

On account of the presence of dissolved salts of various kinds, milk conducts the electric current. Koeppe (3) found the electrical conductivity of cow's milk to be $43.8.\ 10^{-4}$ and that of human milk to be $22.6.\ 10^{-4}$. He concludes therefore that in cow's milk 0.072 and in human milk 0.04 grammolecules (Molen) exist in the ionic condition, or, in other words, that in cow's milk 58 per cent and in human milk 26 per cent of the molecules are dissociated.

The specific heat of milk has been determined by Fleischmann (4). For milk containing 3.17 per cent of fat he finds the specific heat to be 0.9457. This same author also determined the coefficient of expansion of milk by heat and found it between 5° and 15° C. to be greater than that of water. According to Fleischmann (5) milk shows no maximum of density above 1° C.

The viscosity of milk has been determined by Soxhlet (6) using a Reischauer viscosimeter. The following are the ratios of the intervals required for the delivery of the same volume of water and milk at different temperatures:

Temperature	Ratio of water to milk.	Temperature.	Ratio of water to milk.
0° C	100 : 221.1	20° C	100 : 211.7
5° C	100 : 207.7	25° C	100 : 175.9
10° C	100 : 190.6	30° C	100 : 169.0
15° C	100 : 188.7		

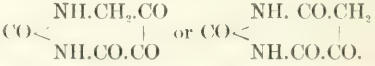
The microscopic examination of milk reveals the presence of great numbers of fat globules, and according to Cohn (7) and also Savage (8) the presence also of leucocytes and streptococci derived from the udder of the cow. (For further information on this subject see article 13 of this bulletin, "The significance of leucocytes and streptococci in milk," by W. W. Miller.)

Lawrence (79) has recently observed an instance of the appearance of typhoid bacilli in the milk of a nursing woman ill with typhoid fever.

With the higher powers of the microscope various forms of bacteria can be distinguished, some of which at least play an important part in the changes which take place when milk is kept for some time at ordinary temperatures. The perfectly fresh milk of carnivorous animals is as a rule acid in reaction. According to Leach (9) the acidity of fresh milk is due to carbon dioxide and acid phosphates, and according to Richmond (10) to mono- and di-phosphates. Human milk and that of herbivora is slightly alkaline and cow's milk has been described as amphoteric; that is, it is alkaline to red litmus, acid to blue litmus. Vogel (11) states that he has never yet found perfectly freshly drawn cow's milk to show a decidedly alkaline reaction to litmus. In the greater number of instances the reaction of freshly drawn milk was either neutral or transiently acid. On standing exposed to the air for some time all forms of milk become more or less acid in reaction in consequence of the conversion of milk sugar into lactic and other acids by the action of various micro-organisms, until finally considerable amounts of acid are produced, which are responsible for the souring and curdling of the milk ordinarily observed.

For further information relative to the reaction of human and cow's milk and for a theoretical explanation of the acidity and alkalinity shown by these milks, see Courant, pp. 349–350.

Milk consists chiefly of water. In addition to this it contains fat, lactose, several proteids (see Halliburton (12)), such as caseinogen, lactalbumin, lactoglobulin, opalisin, and lactomucin, and a number of salts. It also contains certain dissolved gases, such as oxygen, nitrogen, and carbon dioxide. The oxygen and nitrogen are carried into the milk mechanically in the process of milking. Carbon dioxide is present in milk to the extent of 3 or 4 per cent by volume and partly escapes into the air when milk is drawn from the udder. Besides the substances already mentioned still others have been found in milk in small quantities. Among these may be mentioned lecithin (13), cholesterol (14), citric acid (15), lactosin, a new carbohydrate (16), and orotic acid (17). This substance has the composition $C_5H_4O_4N_2.H_2O$, and is believed by its discoverers to have the constitution:



$$CO\!<\!\begin{array}{l} NH.CH_2.CO \\ \qquad\qquad\quad | \\ NH.CO.CO \end{array} \quad \text{or} \quad CO\!<\!\begin{array}{l} NH.\ CO.CH_2 \\ \qquad\qquad\quad | \\ NH.CO.CO. \end{array}$$

Sherman, Berg, Cohen, and Whitman (18) found small amounts of ammonia in fresh milk. According to Trillat and Sauton (19) the presence of ammonia in fresh milk is usually indicative of contamination. According to Schöndorf (20) human milk contains small amounts of urea. Jolles (21) and others have called attention to the relatively large amounts of iron which woman's milk normally contains, and to its influence on the health of the child. Camerer (22) found 21 milligrams of iron oxid in 100 cubic centimeters of human milk from the third to the twelfth day of lactation. According to Jolles and Friedjung (23) the quantity of iron in human milk decreases with bad environment and poor condition of the mother.

In certain diseased conditions milk may contain still other substances not ordinarily present in the milk of healthy animals. For example, Van der Marck (24) has detected bile in the milk of a woman who had developed jaundice after confinement, and Desmoulières and Gautrelet (25) have found that the so-called lipochrome of cow's milk consists almost entirely of urobilin. Still other substances are sometimes acquired by milk either from the food of the animal or from its environment after its removal from the animal. Bordas and Touplain (26) have shown for example that milk rapidly absorbs certain odoriferous substances from the air, and Dombrowski (27) has shown that the odor and flavor of certain seeds and plants are imparted to the milk by feeding with these substances. An excellent example of this is furnished in the case of garlic. According to Rosemann (28) alcohol passes into the milk when adminstered to an animal in large amounts. Similarly Teichert (29) observed that the milk of cows fed 90 per cent "slump" contained fusel oil and that calves fed with such milk died. According to Béchamp (30) even freshly drawn milk contains recognizable amounts of alcohol and acetic acid. Golding and Feilmann (31) detected copper in a certain milk supply, and have shown that in the presence of air milk has the power of dissolving small quantities of this metal.

In addition to the substances already mentioned, normal milk contains a number of enzymes, such as diastase (amylase), galactase, lipase, catalase, peroxidase, reductase, etc. The presence of these ferments serves to distinguish raw from boiled milk. According to Marfan and Gillet (32) milk is not an inactive fluid, but possesses certain properties peculiar to living substances. According to these authors it contains ferments and gives Bordet's reaction (see p. 335), which reaction is not shown by dead material. It also shows Moro's reaction (see p. 335). These specific ferments of milk and its characteristic biochemical reactions will be considered at length under milk enzymes (see pp. 335 to 342).

Woodhead and Mitchell (33) have recently shown that milk also contains opsonins in even greater quantity than blood serum. It also contains alexins and bactericidal substances. According to Brieger (34) and his coworkers, the milk of animals immunized against diphtheria and tetanus contains antitoxins.

A very good idea of the quantities of the several more important substances contained in milk may be obtained from the following schemes compiled by Lucius L. Van Slyke (35) and S. M. Babcock (36):

[Van Slyke.]

- Milk 100
  - Water 87.1
  - Solids 12.9
    - Fat 3.9
    - Solids not fat 9.0
      - Nitrogen compounds 3.2
        - Casein 2.5
        - Albumin 0.7
      - Milk sugar 5.1
      - Ash, salts 0.7
      - 9.0
    - 12.9
  - 100.0
  - Gases
    - Carbon dioxide
    - Nitrogen
    - Oxygen

[Babcock.]

Milk	Constituent	Per cent	Group	Per cent	Class	Per cent	
Butter fat 3.6	Olein		Glycerides of insoluble and nonvolatile acids	3.3	Fat	3.6	Total solids 12.7
	Palmitin						
	Stearin						
	Myristin						
	Butin (trace)						
	Butyrin		Glycerides of soluble and volatile acids	0.3			
	Caproin						
	Caprylin (trace)						
	Caprin (trace)						
Milk serum 96.4	Casein	3.00	Containing nitrogen	3.8	Solids not fat	9.1	
	Albumin	0.60					
	Lactoglobulin, Galactin, Fibrin (trace)	0.20					
		3.80					
	Milk sugar			4.5			
	Citric acid			0.1			
	Potassium oxide	0.175	Ash	0.7			
	Sodium oxide	0.070					
	Calcium oxide	0.160					
100.0	Magnesium oxide	0.017					
	Iron oxide	0.001					
	Sulphur trioxide	0.027					
	Phosphoric pentoxide	0.170					
	Chlorine	0.100					
		0.7		9.1		12.7	
	Water						87.3
							100.0

According to Farmers' Bulletin No. 29, United States Department of Agriculture (37), 1895, cow's milk has the following composition:

					Per cent.
Water	87				87.0
Solids =13		Fat	3.6		3.6
		Solids not fat	9.4	Casein	3.3
				Albumin	0.7
	100		13.0	Milk sugar	4.7
				Ash	0.7
					100.0

Van Slyke (38) gives the following average analysis of cow's milk:

	Water.	Total solids.	Fat	Casein.	Albumin.	Sugar.	Ash.
Average of 5,552 American analyses compiled by the author	87.1	12.9	3.9	2.5	0.7	5.1	0.7
Average cheese factory milk for the season (May to November) in New York state	87.4	12.6	3.75	2.45	0.7	5.0	0.7

The following compilation, according to Leach (39) from Koenig's Chemie der menschlichen Nahrungs- und Genussmittel, gives a very good idea of the composition of human milk and that of a number of different animals:

Number of analyses.	Kind of milk.	Specific gravity.	Water.	Casein.	Albumin.	Total proteids.	Fat.	Milk sugar.	Ash.
800	Cow's milk:								
	Minimum	1.0264	80.32	1.79	0.25	2.07	1.67	2.11	0.35
	Maximum	1.0370	90.32	6.29	1.44	6.40	6.47	6.12	1.21
	Mean	1.0315	87.27	3.02	0.53	3.55	3.64	4.88	0.71
200	Human milk								
	Minimum	1.0270	81.09	0.18	0.32	0.69	1.43	3.88	0.12
	Maximum	1.0326	91.40	1.96	2.36	4.70	6.83	8.34	1.90
	Mean	...	87.41	1.03	1.26	2.29	3.78	6.21	0.31
200	Goat's milk:								
	Minimum	1.0280	82.02	2.44	0.78	.....	3.10	3.26	0.39
	Maximum	1.0360	90.16	3.94	2.01	.....	7.55	5.77	1.06
	Mean	1.0305	85.71	3.20	1.09	4.29	4.78	4.46	0.76
32	Ewe's milk:								
	Minimum	1.0298	74.47	3.59	0.83	.....	2.81	2.76	0.13
	Maximum	1.0385	87.02	5.69	1.77	.....	9.80	7.95	1.72
	Mean	1.0341	80.82	4.97	1.55	6.52	6.86	4.91	0.89
17	Mare's milk:								
	Mean	1.0347	90.78	1.24	0.75	1.99	1.21	5.67	0.35
7	Ass's milk								
	Mean	1.0360	89.64	0.67	1.55	2.22	1.64	5.99	0.51

Bunge (40) gives the following table showing the results of analyses of the milks of a number of different animals:

One hundred parts of milk contain—

	Human.			Dog.	Cat.	Rabbit.	Guinea pig.	Sow.	Elephant.
	I.	II.	III.						
Casein	....	1.2	....	5.2	3.1	....	....	....	....
Albumin	....	0.5	....	1.9	6.4	....	....	....	....
Total proteids	1.7	1.7	1.5	7.1	9.5	15.5	11.2	5.9	3.1
Fat	3.1	3.8	3.3	12.5	3.3	10.5	45.8	6.9	19.6
Sugar	5.9	6.0	6.5	3.5	4.9	2.0	1.3	3.8	8.8
Ash	0.2	0.2	0.3	1.3	0.6	2.6	0.6	1.1	0.7

	Horse.	Ass.	Cow.	Goat.	Sheep.	Reindeer.	Camel.	Llama.	Porpoise.
Casein	1.2	0.7	3.0	3.2	5.0	8.4	....	3.0	....
Albumin	0.8	1.6	0.5	1.1	1.6	2.0	....	0.9	....
Total proteids	2.0	2.2	3.5	4.3	6.5	10.4	4.0	3.9	[a] 7.6
Fat	1.2	1.6	3.7	4.8	6.9	17.1	3.1	3.2	43.8
Sugar	5.7	6.0	4.9	4.5	4.9	2.8	5.6	5.6	....
Ash	0.4	0.5	0.7	0.8	0.9	1.5	0.8	0.8	0.5

[a] Proteids and sugar of milk.

He calls attention to the extreme variability in the composition of the milk of different animals. The large amount of fat contained in some of these milks is certainly very striking. On the other hand, the milks of most of these species show a reasonable similarity so far as the amounts of the several constituents are concerned.

H. Droop Richmond (41) has made a very large number of analyses of milks sold in England. As a rule he found the average composition of the milks produced in that country to be considerably in excess of the legal requirements. The following table will give some idea of the results of his analyses for a number of years:

Year.	Number of milks analyzed.	Total solids.	Fat.	Year.	Number of milks analyzed.	Total solids.	Fat.
1900	13,798	12.57	3.61	1901	15,910	12.68	3.74
1901	13,936	12.63	3.72	1905	14,828	12.70	3.73
1902	12,914	12.73	3.82	1906		12.64	3.71
1903	15,313	12.78	3.83				

This author (42) also gives a single analysis of woman's milk and that of a she ass, which are worthy of record in this connection:

Kind of milk.	Total solids.	Fat.	Sugar.	Proteids.	Ash.	Solids not fat.	Acidity.
Ass's milk	10.27	1.45	5.65	2.09	0.54	(8.82)	0.54
Woman's milk	13.97	5.61	6.98	1.27	0.18	8.36	....

Billitz (43) gives the following results of the analyses of 187,610 specimens of milk produced in Lombardy during the years 1892 to 1902:

Specific gravity	1.0315
Fat	3.55
Solids not fat	8.81

The poorest milk from a herd of 50 cows gave the following numbers:

Specific gravity	1.0306
Fat	2.70
Solids not fat	8.45

The richest milk from a herd of 80 cows gave the following:

Specific gravity	1.0326
Fat	4.10
Solids not fat	9.23

These figures suffice to give an idea of the average composition of milk.

On the other hand cow's milk is liable to extreme variations in composition. For example, Cook and Hills (44) have recorded the following analysis of the milk of a Jersey cow just before she went dry:

Total solids	28.43
Fat	14.67
Solids not fat	13.76
Casein and albumin	9.98
Milk sugar	2.33
Ash	1.44

This milk is remarkable for the large amounts of fat, proteid, and ash, and for the small amount of milk sugar. According to these authors there seems to be no other record of a milk showing more fat than solids not fat. On the other hand Wanters (45) has recorded analyses of several milks showing very small amounts of fat and nonfatty solids:

	Fat	Solids not fat.
(*a*)	1.319 to 2.575	5.031 to 7.635
(*b*)	1.250 to 2.965	6.19 to 8.085

The ash of these milks was abnormally high.

Janke (46) also reports the results of analyses of certain samples of milk supplied the city of Bremen. The samples were taken in the presence of a police officer and are remarkable for the small amounts of total solids and fat which they contained. His results are as follows:

	Total solids.	Fat.
(*a*)	7.71	0.868
(*b*)	6.80	633
(*c*)	8.23	116

Out of another lot of 103 samples analyzed by this chemist (47), the poorest milk had a specific gravity of 1.0275 and contained 9.04 per cent total solids and 1.60 per cent of fat.

The composition and also the yield of milk have been found to vary with the seasons of the year, with the character of the food, with the condition of the animal, and also whether it is fatigued or at work or at rest. It is also subject to some diurnal variation (Richmond (48)). It is also influenced by the addition of certain stimulants and nitrogenous compounds to the food. It also varies in composition during the course of lactation and also at different stages of the same milking. Sherman (49) has shown by monthly analyses extending over two years on a herd of 600 cows that the per cent of proteids in milk and likewise the fat varies with the season, being higher in autumn and winter than in the spring or summer. On the other hand the percentage of lactose remains practically constant throughout the year. Richmond (50) also found the lowest percentage of fat in May and June and the highest during the winter months. On the other hand he found that the geology of the region over which the herd grazed exerted but little influence on the composition of the milk.

Concerning the effect of food on the composition of milk, there seems to be a good deal of difference of opinion among different investigators, some holding that the character of the food exerts a great influence on the character of the milk, others maintaining that this influence is but slight if any. According to Albert and Maercker (51) rations rich in fat cause a decided increase in the fat of the milk. If this however be continued for long intervals the fat falls to its original amount with the poorer rations. Rhodin (52) found that emulsified oils cause an increase in the amount of fat, followed by a return to the normal amount. These observations were confirmed by Bartlet (53).

Gogitidse (54) found that by feeding sheep with linseed oil the fat of the milk could be made to contain as much as 33 per cent of linseed fat. Hills (55) observed that the addition of cotton seed, maize, or linseed oils to the food of cattle tends to increase the yield of milk per unit of dry matter fed. With cotton-seed oil there seemed to be a fairly permanent increase of 0.2 to 0.3 per cent of fat in the milk. On the other hand maize and linseed oils, when given as a regular diet, while causing a marked increase in the fat at first, seemed to lower the percentage of fat in the later stages of the experiment. Essentially similar results have been obtained by V. Henriques and Hansen (56). Sebelien (57) has found that the effect of feeding whale meal was to increase the yield of milk 6 per cent during the period when it was given. There was no after effect. The absolute amount of fat was increased during the first period of whale-meal feeding, but sank during the last period to the amount produced in the preliminary period. The percentage of fat was not altered by the whale meal when this was given as additional food, but was lowered when an extra quantity of it was given. Wing (58) found that the addition of fat to the fodder neither increased the quantity of milk nor the amount of fat which it contained. Morgen, Beger, Fingerling, Doll, Hancke, Sieglin, and Zielstorff (59) working together, have shown as the result of an extensive series of investigations on the effect of foods and food fat on the production of milk and milk fat in sheep and goats that food almost free from fat maintained the animals in healthy condition and increased the live weight of the animal, but that such foods were unsuitable for milk production. Food fat in small quantities, 0.5 to 1 gram weight per kilo of the animal was found to promote the production of milk fat. They proved further that so far as their effect on milk production and the increase of fat in milk is concerned, stimulants are only desirable in certain cases. These investigations have been further extended by these observers working together or alone. For example, Morgen, Beger, and Fingerling (60), as the result of studies extending over six years, have reached the conclusion that of all foods, fat alone exerts a specific action on the production of milk fat, proteids and carbohydrates exerting no such action, and that within certain limits fat is the most suitable of all foods for milk-fat production. In this same connection, Fingerling (61) has shown that the replacement of food deficient in fat (barley meal) by one containing more fat (rice meal) increased both the absolute amount and the percentage of fat in the milk. From a study of the influence of stimulants on the consumption and digestibility of food and the secretion of milk he (62) has also arrived at the conclusion that when added to foods entirely free from stimulants the effect of the stimulant is to increase the consumption of food and the yield of milk and milk constituents. When however stimulants are added to foodstuffs already containing such substances they are without effect on the yield of milk. He concluded therefore that they are of use only in special cases, as, for example, when cattle are fed with hay. In such cases the addition of such materials to the food as fenugreek, anise, and caraway seed is to be recommended. According to Temesvary (63) beer increases the amount of milk fat. Morgen, Beger, and Fingerling (64) have also investigated the influence of fat and other substances on milk production when fed in connection with a scanty basal meal. They have observed an increase in the yield of milk and an increase in the percentage of fat amounting to 0.14 per cent when such quantities of fat were added to the food. The addition of large quantities of fat to the food caused a further increase in the yield of milk, but was found to vary in its effect on milk-fat production, sometimes causing an increase, sometimes a decrease. Caspari (65) has shown that iodized fats appear in the milk even though the food be poor in fats and rich in carbohydrates. He therefore concludes that some of the fat of milk comes from the fat of the food. Later (66) he showed that when iodocasein and iodoalbumin are fed to an animal no traces of iodized fats appear in the milk. On the other hand there are those who hold that the addition of fat to food does not increase the quantity of fat in milk and that there is no direct migration at least of the fat of the food to the milk. Such a conclusion was arrived at by Einecke (67) from his experiments with goats. With liberal comprehensive rations the yield of milk and fat depends, according to this observer, on the individuality of the animal. The milk from cows grazing off the poor, dried-up grass on the plateau of Sétif, in Algeria, has been compared by Malméjac (68) with that of cows fed on rich forage with the following results:

	Poor dry grass.	Rich forage.
Total solids	11.62 to 14.25	13.76 to 14.90
Fat	3.33 to 3.50	4.05 to 4.90
Lactose	3.13 to 4.46	3.33 to 4.54
Proteids	4.53 to 5.64	4.47 to 5.55
Ash	0.60 to 0.90	0.82 to 0.93

Except for the proteids, the differences in composition are obviously in favor of the milk produced on the richer diet. Woll (69) observed that as a food for milch cows silage increases the yield of milk and butter 3 per cent over that produced with maize fodder when the area of land required to produce the two foods is taken into account. Some studies have also been made of the effects of certain definite nitrogen and phosphorus compounds on the production of milk and milk fat. Morgen, Berger, and Fingerling (60) have investigated the effect of adding lecithin to food. This substance seemed to increase the yield of milk and also the live weight of the animal. It was found, however, to be favorable to the production of milk fat only when it was fed in conjunction with other foods deficient in fat. Pfeiffer, Einecke, and Schneider (70) have shown that asparagin when substituted for proteids, along with cane sugar, caused no diminution in the yield of milk, in fact in some instances it seemed to cause an increase, but the amount and percentage of fat in milk was diminished. The feeding of this compound also acted unfavorably on the increase in live weight, and caused a reduction in the percentage of proteids and dry matter in the milk. Morgen, Beger, and Westhauser (71) have reached the conclusion that amino compounds can not take the place of proteids in milk production, but that they exert a greater effect than carbohydrates.

It has been observed that the actual yield of milk diminishes in the later period of lactation. According to Trunz (72), however, the specific gravity of the milk, and most of the solids, including the proteids, are relatively increased, while the proportion of albumin to casein remains remarkably constant throughout the entire period of lactation. This same investigator (73) has also made an exhaustive study of the mineral constituents of cow's milk and their variation during the period of lactation with the result that he has found considerable variation in the ash contents during the lactation period and that the total quantity of ash varies from time to time throughout the period, being as a rule less during the spring and summer months than during the autumn and winter months.

Hardy (74) claims to have shown that the milk of a given cow varies in composition at the different stages of milking. Thus taking the milk in quantities of one-half liter at a time the milk of one cow gave the following successive numbers for fat: 2.2, 2.9, 3.5, 3.75, 3.8, and 4.65 per cent. The solids rose from 10.52 to 12.70 and the ash from 0.74 to 0.75 per cent. The composition of the milk serum was found to remain the same throughout the milking.

On the other hand Ackermann (75) claims that the conclusion that the fat in milk increases regularly during the process of milking, as this is ordinarily carried out, is incorrect. He has found, however, that by milking the teats singly or in pairs the fat did show an increase up to a maximum at the end of the milking and that on drawing the milk from the second pair of teats the quantity of fat was slightly more at the commencement than that given by the first pair and rose at the end of the milking to a higher maximum. The increase is probably due to a mechanical or physiological stimulus.

The effect of work and fatigue on the quantity and quality of the milk has also been studied by several observers. Hills's (76) results would seem to show that there is a slight falling off in the quantity of milk produced as a result of fatigue, 122.5 pounds against 131.4 pounds after rest. The total solids and the fat were found to be slightly higher during the period of fatigue than after rest. Dornic (77) also has shown that the yield of milk is diminished slightly as the result of work. The dry matter and the amount of acid were slightly increased. It was further observed by this investigator that work exerts a harmful influence on the quality of the milk, especially on its keeping qualities. For example it was found in the case of a certain cow that ordinarily her milk curdled when the acidity reached 70°–75°, whereas the milk of the same cow when fatigued by work, curdled when the acidity reached 45°. According to Moerman (78) also, work lessens the amount of milk secreted and raises the proportion of solids. The differences, however, in the quantity and quality of the milk in all of these investigations were only slight, indeed in some instances the results obtained were not very definite.

PART II.—CHANGES IN THE COMPOSITION OF MILK.

On account of the milk sugar and proteids which milk contains, it is an exceedingly unstable liquid. When first drawn from the cow, it has a characteristic odor and a sweet taste. Even in the perfectly fresh state, it reacts acid to phenolphthalein. The acidity of fresh milk is due primarily to carbonic acid and acid phosphates and also in part to dicalcium caseinogenate. According to Thörner (1) the acidity of fresh milk varies between 12 and 16 degrees. According to Richmond (2) it has an acidity of 20 degrees. On standing exposed to the air for some time it gradually loses its sweet taste. The sugar of milk is gradually transformed into lactic acid through the action of bacteria. The milk becomes sour to the taste and ultimately clots or curdles as the result of the precipitation of the caseinogen by the combined action of acids and soluble calcium salts. Stokes (3) gives figures and tests to show that milk having an average acidity of 44 degrees, corresponding to 0.396 per cent of lactic acid, tastes sour. According to Richmond (2) milk tastes sour when the acidity reaches 45 degrees, corresponding to 0.405 per cent lactic acid, and when it has an acidity of 85 degrees, equivalent to 0.765 per cent of lactic acid, it curdles at ordinary temperatures.

Under certain conditions, milk may also develop rancid and cheesy odors which render it quite disagreeable.

The principal changes occurring in milk are those produced by

(1) The action of heat and acids.
(2) The action of milk enzymes.
(3) The action of the digestive enzymes.
(4) Bacteria and various other micro-organisms.

PART II. (1) CHANGES IN MILK PRODUCED BY THE ACTION OF HEAT AND ACIDS.

When milk is heated a film or skin forms on the surface, which, according to Jamison and Hertz (4), is due to the drying and coagulation of a part of the proteids which the milk contains. They have shown that such a skin may be formed on the surface of any albuminous solution containing fat or paraffin. Rettger (5) also has arrived at the conclusion that its formation is dependent on the presence of proteid. This proteid is caseinogen. Surface evaporation and the presence of fat facilitate its formation although neither is absolutely essential. According to Harris (6), also, the scum of boiling milk consists very largely of caseinogen. It is also well known that certain changes occur in the odor and taste of milk as the result of boiling. These changes seem to be due to the partial decomposition of certain of the proteids with the liberation of a volatile sulphide, probably hydrogen sulphide. That such is the case has been proven by Rettger (7), and also by Franz Utz (8). According to the former, when milk is heated to 85° C., a volatile substance, probably hydrogen sulphide, is liberated. The amount of this, though small, suffices to blacken lead acetate paper and to decolorize dilute solutions of potassium permanganate. He found that alkalis and alkali phosphates accelerate the formation of the sulphide, whereas acids and acid phosphates retard this change. According to this author this change is believed to indicate proteid decomposition, and may partly account for what some observers describe as the injurious effect of heating milk. These observations have been confirmed by Utz (8), who was able to recognize the hydrogen sulphide resulting from the boiling of milk by lead acetate paper and also by Ganassini's reagent

When milk is boiled there seems also to be a partial fixation of the calcium salts which it ordinarily contains. These are probably partially precipitated in the form of tricalcium phosphate. This would account for the fact that the coagulation of milk by rennin takes place more slowly in boiled milk than in unboiled milk. (See p. 332.) In this connection Wassermann and Schütze (9) have pointed out that cooked milk is not coagulated by lactoserum. According to P. T. Müller (10) the fact that cooked milk can not be coagulated by lactoserum is in some way associated with a diminution in the quantity of soluble calcium salts contained in the milk, this diminution having been caused by the action of heat. On the other hand, both Moro and Müller (10) have observed that certain milks do not show any diminution in coagulability by lactoserum after boiling. According to Müller (10) this is to be attributed to the large amount of soluble calcium salts present in the milk of certain particular localities, and in this connection he has observed that the coagulability by lactoserum may be restored to boiled milk by the addition of soluble calcium salts.

Hammarsten observed that milk curdles when it is heated to 130° to 150° C. (see p. 341). Cazeneuve and Haddon (11) observed that milk which had been coagulated at 130° C. became very acid. According to these observers it then contained formic acid. They also reached the conclusion that the discoloration and coagulation of milk by heat is due to the oxidation of lactose in the presence of the alkaline salts of the milk, one product of the oxidation consisting of formic acid, which, like other acids, precipitates the caseinogen. The latter undergoes no further change except that it is discolored by the products of the decomposition of lactose.

Bruno Bardach (12) has also studied the coagulation of milk by heat. He found that about twelve hours' heating at 100° C. was required in order to coagulate perfectly fresh milk, whereas at 150° C. it coagulates in three minutes, and at 130° C. in one hour. He found only the merest traces of formic acid to be formed at 130° C. He concludes from his study of the subject that the coagulation of milk by heat is a complex process; that it is brought about by the action at the high temperature of the small quantities of acid which are formed from the lactose, and which ordinarily are powerless to coagulate the original unchanged casein (caseinogen), and that it is only after the casein (caseinogen) has been changed by the action of heat that such small amounts of acid can cause its coagulation.

The part played by calcium salts in the acid coagulation of milk has been studied by Loevenhart (13). According to this author the very small quantities of acid required to effect the coagulation of milk at temperatures at or below boiling accomplish this change by rendering the calcium salts normally present in milk available for the coagulation of the caseinogen. Therefore the temperature at which a given specimen of slightly sour milk will coagulate on heating depends partly upon the degree of acidity and also upon the nature and amount of the calcium salts present in the milk.

Von Soxhlet (14) has also recently investigated the coagulation which occurs on boiling faintly acid milk. He observed that at the commencement of the souring of milk boiling causes a coagulum to form. This occurs when only one-eighth of the amount of acid necessary to produce coagulation at ordinary temperatures is present. It depends, according to this author, on the formation of an insoluble compound of caseinogen with soluble calcium salts, the acid first produced forming monocalcium phosphate from the dicalcium phosphate present in the fresh milk.

The fact that milk occasionally curdles in the pasteurizing apparatus during pasteurization makes the accumulation of data bearing on this particular phase of the subject a matter of considerable importance. During our recent investigations of the Washington milk supply we incidentally made a number of observations on the coagulation of slightly sour milk at or below boiling. The results of these observations, aranged in the order of diminishing acidity, are given in the following table:

No. of sample.	Acidity (per cent).	Temperature (° C.).	Time of heating (minutes).	Curdled = +; not curdled = −.
1	0.711	65	*0	+
2	.594	65	1	+
3	.576	65	2	+
4	.567	65	1	+
5	.554	60	2	+
6	.531	65–67	2	+
7	.513	65	2	+
8	.478	60	5	+
9	.450	65	1½	+
10	.441	66	1	+
11	.387	65	5	+
12	.351	65–67	2	+
13	.351	65	5	+
14	.351	65	5	+
15	.342	78.5	2	+
16	.342	66	5	
17	.315	70	10	+
18	.315	70	5	
19	.315	65	5	
20	.306	75	3	+
21	.306	65	5	
22	.288	70	5	
23	.261	65–74	5	
24	.252	100	1	−
25	.252	70	5	
26	.243	100	1	
27	.243	72–74	10	
28	.243	65	10	−
29	.234	65	5	
30	.225	65–67	2	+
31	.198	65	5	
32	.180	65	5	

* Immediately.

It will be seen from our results that of those samples of milk which coagulated on heating, sample 30 contained the smallest amount of acid at the time of coagulation, viz, 0.225 per cent. This sample curdled in two minutes at 65°–67° C. It will be observed that milks containing from 0.306 to 0.315 per cent of acid curdled at temperatures varying from 65°–78.5° C., in from two to ten minutes, and that as a general rule those milks are most easily coagulated by heat which have the highest acidity. On the other hand, while one of the samples having an acidity of only 0.225 per cent curdled at 65°–67° C., in two minutes, another sample containing 0.243 per cent of acid did not curdle even on boiling. Thörner (15) found the acidity necessary to coagulate milk on heating to be 0.207 per cent. On the other hand, Rideal (16) found the amount of acid required to effect the coagulation to be somewhat higher than this. According to him the tendency to coagulate is not very marked even when the acidity is as high as 0.23 per cent. Rideal's observations agree very nearly with our own.

According to Stokes (3) milk which has an acidity somewhat less than 0.3 per cent of lactic acid will coagulate on boiling. He records the fact, however, that 3 samples of milk containing as much as 0.54 per cent of lactic acid did not coagulate on boiling.

Richmond (2) has been able to confirm Stokes's results almost absolutely. He found fresh milk to have an acidity of 20 degrees, corresponding to 0.18 per cent lactic acid. According to him milk curdles on boiling when it has an acidity of 33 degrees, corresponding to 0.297 per cent of lactic acid.

Revis and Payne (17) have shown that at the moment when the caseinogen is precipitated the calcium triphosphate has been eliminated, and that the combination of caseinogen with lactic acid has reached a maximum.

It is evident therefore from our results and those obtained by other observers that the coagulation of milk is dependent on several factors, among which are: time, temperature, degree of acidity, quantity and nature of the calcium salts, etc.; and that in order to avoid accidents resulting from curdling in the pasteurization of milk the only safe rule to follow is to determine the effect of heat on small samples of the milk which it is proposed to pasteurize, or better still, to pasteurize the milk as soon as it is drawn from the cow.

Another important change in milk effected by heat is the destruction of the bacteria and other micro-organisms normally present in fresh milk, including of course those pathogenic forms which frequently gain access to milk and cause the spread of infections through this medium or which give rise directly to highly poisonous substances. For obvious reasons therefore this phase of the subject, namely, the pasteurization of milk, has received a great deal of attention during recent years at the hands of dairymen and sanitarians. It is foreign to the immediate scope of this communication, however, to enter upon a discussion of this subject. Suffice it to say in this connection that pasteurization either checks or hinders those changes which occur in milk as the result of the life and growth therein of micro-organisms, and affords more or less adequate protection against the spread of microbic diseases through the medium of milk. According to Pasteur (18) milk can be sterilized by heating it to about 110° C. and Duclaux kept milk five years by heating it to 120° C. and preserving it in vessels which had been exhausted of air.

Effect of Heat on Milk Enzymes.

Another less obvious change in milk which is brought about by the action of heat is the destruction of the enzymes normally present in fresh milk. Like all enzymes those contained in fresh milk are destroyed by boiling or by heating the milk above certain temperatures. On account of the important bearing which the thermal death point of milk ferments may have on the pasteurization of milk the following data relative to this subject are here submitted:

Galactase.—According to Babcock and Russell (19), the destruction temperature of galactase, the proteolytic enzyme of milk, is somewhat higher than the thermal death point of trypsin. So far as its proteolytic activity is concerned, these observers found it to be weakened by heating for ten minutes at 76°–80° C. That such is the case may be seen from the following table, in which is shown the percentage of soluble nitrogen present at the end of fifty-three days in milks which had been heated for ten minutes at the temperature indicated.

TABLE I.

Temperature to which the galactase was heated for 10 minutes before being allowed to act on the milk.	Per cent of soluble nitrogen after 53 days at 37° C.[a]
78—80° C.	0.05
76	.05
71	.12
65	.20
60	.20

[a] The soluble nitrogen originally present in the milk used in these experiments was found to be 0.05 per cent.

Similar tests were made upon the proteolytic activity of galactase towards gelatin, using Fermi's (20) method. Equal amounts of the galactase solution were poured over the surface of carbolized gelatin contained in test tubes and kept under observation seven weeks. The results of this series of experiments is given in Table II.

TABLE II.

[+ + =rapid digestion; + =digestion by galactase; – =no digestion.]

Temperature (°C.).	Reaction of the galactase solution.			
	Alkaline.		Neutral.	Acid N/10.
	N/10.	N/20.		
65	[illegible]	[illegible]	[illegible]	[illegible]
70	[illegible]	[illegible]	[illegible]	[illegible]
75	[illegible]	[illegible]	[illegible]	[illegible]
80	[illegible]	[illegible]	[illegible]	[illegible]
Control	[illegible]	[illegible]	[illegible]	[illegible]

Hence so far as its power to digest gelatin is concerned the activity of galactase is destroyed in alkaline solution by ten minutes' exposure to temperatures of 65° C. in neutral solutions at 70° C. and in acid solutions at a temperature of 65° C.

Babcock and Russell (19) have also employed the power on the part of galactase to accelerate oxidations by hydrogen peroxide as a test for the presence of the ferment, and have made use of this test as a means of determining the destruction temperature of galactase. This we now know is merely a test and measure of the peroxidase activity of the solution and not a test or measure of the activity of galactase at all. Indeed, as has been shown subsequently to the work of Babcock and Russell on this subject, galactase as prepared from separator slime is not a pure enzyme, but a mixture of enzymes. Thus Wender (21) points out that the galactase of Babcock and Russell consists of milk trypsin or galactase proper, milk catalase, and milk peroxidase. According to Wender, the trypsin of milk becomes inactive at 76° C.

The use of V. Storch's tests (see p. 333), viz. with an iodide and starch or p-phenylene-diamine and a few drops of hydrogen peroxide as a means of determining the effect of high temperature on the activity of galactase, as employed by Babcock and Russell, is therefore chiefly interesting as throwing light on the stability of milk peroxidase under the conditions employed in their work. Their results are given in Table III.

TABLE III.

[+—color reaction; ±—doubtful reaction; −no color reaction.]

Temperature (°C.).	Time.	Alkaline.		Neutral to litmus.	Acid N/10.
		N/10.	N/25.		
65	10	+	+	+	+
	30	+	+	+	+
	60	+	+	+	−
70	10	+	+	+	+
	30	+	+	+	−
	60	+	+	+	
75	10	+	+	+	
	30	+	+	+	
	60	+	+	+	
80	10	+	+	+	
	30	+	+	−	
	60	−			

Babcock and Russell (19) conclude therefore from their experiments that galactase is more stable in alkaline or neutral solution, and that it shows a close resemblance to trypsin, but is less sensitive to acids than the latter ferment. The heat boundary of its activity is influenced by the chemical reaction of the solution in which it is present, being lower in acid than in neutral or alkaline solution. When galactase that has been heated to 70° C. for ten minutes is added to milk, the digestion is slowed, and heating to 76° C. for ten minutes entirely destroys its digestive powers. In the Fermi (19) gelatin tests no digestion took place with specimens of the ferment that had been heated to 65° C., whereas with the unheated controls, the gelatin liquefied.

Von Freudenreich (22) has confirmed the observations of Babcock and Russell on galactase. According to this observer a temperature of 75° C. for half an hour causes a falling off in the proteolytic activity of this enzyme. On the other hand, he found an exposure to 60° C. for half an hour to scarcely weaken it to a noticeable degree. According to Hippius (23) the proteolytic ferments of milk can withstand an exposure to 60° C. for one hour and an exposure to 65° C. for half an hour.

Milk amylase, according to this author, can withstand a temperature of 60° for one hour, but is destroyed by heating above 75° C.

Milk lipase. Gillet (24) has found that milk lipase is destroyed at 65° C. According to Hippius (23) the lipase of woman's milk can withstand a temperature of 60° for one hour and a short exposure to 62°, but is weakened at 63° C., and is destroyed at 64° C.

The salol-splitting ferment (Nobécourt and Merklen) is, according to Hippius (23), destroyed above 65° C. However, the existence of this ferment is doubtful. (See p. 344.)

Hougardy (25) has found that the activity of lactokinase is greatly enfeebled by heating for twenty minutes at 75° C. and practically destroyed by heating for half an hour at this temperature.

The oxidizing ferments of milk.—While our knowledge of the soluble ferments is of too recent date for an exact understanding of their significance and powers of resistance, the well-known reactions of the oxidases have furnished us with a useful criterion for distinguishing between raw (living) and heated milk (Zelinski 26).

According to Marfan (see Zelinski (26)) the oxidases of milk are destroyed at 79° C. According to Hippius (23) they can withstand a temperature of 60° C. to 65° C., but are destroyed by a short exposure to 76° C.

Peroxidases.—According to Wender (21) the peroxidase of milk becomes inactive at 83° C. According to Schardinger (27), using methylene blue as a reagent, the critical temperature of the milk peroxidases is 80° C. With Arnold's guaiacum reagent milks heated to 80°, according to Ostertag, failed to show the reaction (see Glage (28)). Franz Utz (29), using Schaffer's (30) reagent, found that milk heated for a short time at 90° C. or a long time at 70° C. failed to show any reaction. Rullmann (31) gives the following data bearing on the stability of the peroxidases of milk, as shown by Storch's p-phenylene-diamine reagent:

1. Raw milk, not heated.	Deep greenish blue at once.
2. Milk heated one hour, 68°–69° C.	
3. Milk heated one-half hour, 72° C.	No coloration after ten minutes.
4. Milk heated one-half hour, 90° C.	
5. Milk boiled one-half hour.	

V. Storch (32), employing his own reagent, found that exposure of the milk to a temperature of 75° C. for two minutes prevented the reaction. Freeman (33), working with Storch's reagent, found a temperature of 78° C. to destroy the peroxidase of milk. Du Roi and Kohler (34) have employed hydrogen peroxide and the potassium-iodide-starch reagent and have found 80° C. to be the limit of the reaction. Weber (35) recommends Arnold's (36) reagent (guaiacum in acetone), and finds the temperature limit of the reaction to be about 80° C. Franz Utz (37), using a solution of ursol D, finds that milks which have been heated to 80° or above fail to give the peroxidase reaction. According to Rullmann (38) practically all bacteria are destroyed in cow's milk by heating it for one hour to 68°–69° C.; whereas, using p-phenylene-diamine hydrochloride, which he found to be the most delicate reagent for the peroxidase, he found that the latter was not destroyed unless the temperature exceeded 70° C. According to Van Itallie (39) the peroxidases of milk are not destroyed below 80° C., and milk sold as pasteurized milk gave the test with paraphenylene diamine and hydrogen peroxide.[a] Bruere (40) observed that milk which had been pasteurized at 80° C., or boiled, failed to show the peroxidase reaction with the guaiacol reagent. Dupouy (41), using freshly prepared paraphenylene-diamine and hydrogen peroxide, found that unheated milks gave a blue color, whereas those heated above 79° C. gave no color with this reagent.

Douglas (42), using ortol as a reagent, found that heating for five minutes at 75° C., or one minute at 80° C., destroys the peroxidase of milk. Marfan and Gillet (43), using guaiacol as a reagent for the peroxidases in milk, found the ferments still active at 70° C. They were weakened at 75° C., however, and were destroyed at 78° C. to 79° C. Macadie (44) found that nearly all photographic developers, together with small amounts of hydrogen peroxide, give characteristic color reactions with milk which has not been heated above 75° C. Wilkinson and Peters (45), using benzidine and hydrogen peroxide, failed to obtain the peroxidase reaction with milks which had been heated to 78° C. or higher.

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[a] We have also found pasteurized milk to show the peroxidase reaction, with guaiacum, p-phenylene-diamine and also with phenolphthalin.

Using an acetone solution of guaiacum (purified according to Portier (46) and dilute hydrogen peroxide) we made the following observations on the resistance of the milk peroxidases to heat:

Temperature (°C.).	Time of exposure.	Color with reagent.
70	5 minutes	Dark blue.
70	8 minutes	Do.
72	10 minutes	Do.
70	15 minutes	Dark blue; slow in developing.
70	60 minutes	None.
60	15 minutes	Dark blue.
60	30 minutes	Do.
60	60 minutes	Dark blue; somewhat slow in developing.

It is evident, therefore, that while short exposures of milk to a temperature of 60° C. are apparently without effect on the activity of the peroxidases, an hour's exposure to this temperature renders them somewhat less active than in unheated milk, and an exposure of one hour to a temperature of 70° C. destroys them.

It was also observed that milk just brought to 75° C. and 80° C. no longer gives the blue color with the guaiacum reagent. An examination of curdled milk with this reagent revealed the presence of the peroxidase chiefly in the whey and apparently in unaltered condition so far as activity is concerned.

[Since the foregoing was written the whole subject of the peroxidase reaction of milk has been thoroughly investigated by Kastle and Porch (47). These observers succeeded in showing, first, that the power of milk to induce the oxidation of phenolphthalin and other leuco-compounds by hydrogen peroxide is greatly intensified by certain substances of the phenol type; and that by the use of these peroxidase-accelerators, phenolphthalin, guaiacum, and paraphenylene-diamine can all be used to advantage and with certainty as peroxidase reagents for milk; second, that the fresh milks of different cows of the same herd exhibit considerable differences in peroxidase activity; third, that by means of the peroxidase reaction thus modified it is possible to distinguish between raw and cooked milk or between raw milk and that which has been sterilized at a temperature of 70° C. or higher for short intervals, and that while milks which have been heated to 70° C. for one hour, or to 75° C. for twenty minutes, no longer show the peroxidase reaction, this reaction is not diminished in intensity, but, if anything, somewhat increased, by heating the milk to 60° C. for twenty minutes. It is evident, therefore, that the pasteurization of milk at 60° C. for twenty minutes, as recommended by Rosenau, does not destroy the biological properties of milk, at least so far as we are able to judge from the peroxidase reaction.]

Catalase.—Less is known concerning milk catalase than is known of the peroxidases. It would seem, however, that it is perhaps more sensitive to heat than the peroxidases. Van Itallie (48) has shown that cow's milk is unable to decompose hydrogen peroxide after an exposure of one-half hour to 63° C. On the other hand, human milk still retains this power after a similar exposure. According to Jolles (49), heating to 75° C. destroys the catalases of woman's milk completely. Wender (21) observed that catalase prepared from separator slime (see galactase) becomes inactive at 80° C.

Other biological properties.—According to Hippius (23) the bactericidal power of milk is retained even after long heating at 60°–65° C., but is weakened by a short exposure to 85° C., whereas according to Behring (50) this property of milk is lost after an hour's heating to 60° C.

The alexins of milk, according to Behring, are affected by heat in exactly the same way and to the same extent as the antibacterial substances. Lane-Claypon (51) has shown that on adding blood corpuscles to milk the cream picks them up and carries them to the top, and that this property of milk is destroyed by heating it to 70° C. for a few minutes. It is evident, therefore, from these considerations, that the thermal death point of the milk ferments is dependent on a number of conditions besides the temperature itself. Chief among these are time and the reaction of the medium. As a rule, the soluble ferments can withstand somewhat higher temperatures in neutral or slightly alkaline solution than in acid solution. Acidity and temperature naturally augment the destructive powers of each other toward the enzymes. This accounts for the apparent discrepancies existing among such observations. In general, it may be said, however, that the milk ferments, most of them at least, can withstand a temperature ranging from 60°–65° C. for some time, without material injury. Between 65° C. and 70° C. most of them are weakened in their activities, and between 70° C. and 80° C. all of them are destroyed, even after relatively short exposures.

According to Raudnitz (52) all the ferments of milk are destroyed between 75° C. and 90° C. (See also Tjaden, Koske, and Hertel (53), and also E. Weber (54), for further information bearing on this point.)

The digestibility of raw and heated milk.—In this connection the effect of heat on the digestibility of milk proteids has been the subject of considerable investigation. According to Kerr (55) milk is to be looked upon as consisting of living cells suspended in serum, the former consisting of fat cells and nucleated cells of the nature of white blood corpuscles. (As a matter of fact it has been definitely proven that leucocytes do actually occur in milk—see p. 191.) According to Kerr, when milk is ingested, these living elements are at once absorbed without any preliminary digestion, and are utilized directly in the building up of the tissues. The effect of boiling therefore is obviously to kill all of the living cells of the milk, and to coagulate certain of the albuminoid constituents. The result of the boiling therefore is that all of the constituents of the milk must be digested before they can be absorbed into the system. Hence there is a distinct loss of utility in the milk as the result of boiling. He goes on to say further that it has been observed by many medical practitioners that there is a very distinctly lowered vitality among infants which are fed on boiled milk, the process of absorption being delayed and the quantity of milk required for the nourishment of the child being greater than when fresh milk is used.

While this is doubtless an extreme view to take of the matter, there are quite a number of observers who believe that the proteids of milk are considerably altered by boiling. Thus according to Halliburton (56) the milk proteids are rendered somewhat more difficult of digestion as the result of heating. Rubner (57) has observed that even a short heating at 100° C. coagulates the lactalbumin, an observation which has been confirmed by Middleton (58). De Jager (59) has also arrived at the conclusion that the digestibility of milk diminishes with cooking, and also that caseinogen is more readily digestible than casein and that infants stand raw milk better than cooked.

In this connection it has been observed by Lörcher (60) that cooked milk coagulates with rennin more slowly than uncooked milk. This effect is noted even at temperatures of 80°–90° C. This is shown by the following:

Ten cubic centimeters of milk was heated to the following temperatures for five minutes, then cooled to 35° C., and 0.5 cubic centimeter of rennin solution added, and the time required for coagulation noted. The following results were obtained:

Temperature (°C.).	Time required for coagulation (minutes).	Temperature (°C.).	Time required for coagulation (minutes).
50	4¼	80	6½
60	4¼	90	8½
70	4¼	100	9¼

The probable explanation of the retardation of the rennin coagulation resulting from the heating of milk is that the calcium salts are partly rendered insoluble, probably through conversion into tricalcium phosphate, so that even the mineral constituents of milk seem to be somewhat altered by boiling.

On the other hand there are those, among them Forbes Ross (61), who contend that heat exerts no deleterious effect on the digestibility of milk, and that the feeding of infants with boiled milk is not in any way responsible for scurvy or rickets, but in reality is a wise precaution against infantile diarrhea and other bacterial diseases. Similarly Tjaden, Koske, and Hertel (53) claim that by rapidly heating the milk to 90° C., with constant shaking, the chemical and physical properties of the milk are in no wise altered nor is the value of the milk as a food in any way impaired.

These fundamental differences of opinion regarding the effect of heat on the digestibility of milk can therefore only be settled by further investigations along this line.

By way of comparison there are given in the subjoined table certain data pertaining to the destruction by heat of enzymes in general.

This table has been compiled from observations recorded by Green (62), Oppenheimer (63), and others.

Name of ferment.	Temperature at which destroyed (°C.).	Name of ferment.	Temperature at which destroyed (°C.).
Bromelin	Weakened at 70.	Maltase	55.
Caroubinase	80, weakened at 70.	Myrosin	81–85.
Cytase	60.	Oenoxydase	72.
Dextrinase	75.	Papain	82.5.
Diastase (plant)	80.	Pepsin	55–57.
Diastase (saliva)	65–70, slowly at 58.	Rennin	70, neutral; 63, faintly acid.
Emulsin	70.		
Enterokinase	65 (slowly).	Trehalase	64.
Invertase	70, very slowly at 45–50.	Trypsin	75–80.
Laccase	60–63.	Thrombase	70 (Schmidt).
Lipase	72 (Hanriot), 65–70 (Kastle&Loevenhart).	Tyrosinase	55.
		Urease	75–80.

P. T. Müller (64) gives the following data bearing on the subject of the resistance of the several ferments to heat: Hemodiastase (Hahn) is not destroyed at 55° C. It is weakened by heating to 66° C. for half an hour and at 65°-70° C. is destroyed. Parachymosin (Bang) withstands a temperature of 75° C. for some time. Papain is weakened at 75° C. and destroyed at 82.5° C. The oxidizing ferments (Abelous and Biarnès) increase in activity from 0° C. to 60° C. At 80° C. they are still active and are first destroyed at 100° C. Lactoserum (P. T. Müller) is rendered inactive by heating for half an hour, at 70°-75° C. So far then as the influence of temperature on their activity is concerned it is evident that the milk enzymes are much like enzymes from other sources. Indeed they are all very much alike and all of this great group of substances stand in such intimate and close relation to the vital activities of the cell that all of those conditions and influences which tend to destroy the one tend also to destroy the other. (See also Kastle (65) "On the Vital Activity of the Enzymes.") All of the bacteria of milk can not be destroyed therefore without at least diminishing the activity of the milk enzymes or perhaps destroying them altogether and the enzymes can not be destroyed without rendering the milk sterile.

Similar conclusions have been reached by Marfan (66). According to this author the enzymes in general can not withstand a temperature higher than 70° C., so that by heating milk to deprive it of its bacteria, we also deprive it of those ferments which probably exercise a favorable influence on nutrition. He is of the opinion, however, that this is not sufficient ground for doing away with the practice of sterilizing milk by heat. In this connection Rosenau has recently made the important observation that the pathogenic bacteria commonly found in milk are either killed or rendered inert by exposure to 60° C. for twenty minutes, see Hyg. Lab. Bull. No. 42.

Bokorny (67) has also pointed out that between protoplasm and the enzymes there are certain striking similarities. Toward temperature, he says, it has long been known that the enzymes conduct themselves like protoplasm. His table, setting forth these analogies so far as the influence of temperature and light are concerned, is given in the following:

Name of protoplasm or enzyme	Action of temperature, light, etc.
Protoplasm of bacteria and fungi	Nageli found the spores of bacillus subtilis could be heated 11 hours in boiling water without showing the slightest damage. In the vegetative state this organism is largely destroyed by heating to 55°–60° C.
	Light tends to destroy many bacteria.
Yeast	Direct sunlight destroys by long exposure.
	A temperature of 25°–30° C. best for the development of yeast.
	Young vegetative yeast-cells are killed at 50°–60° C., spores at 60°–65° C. In the dry state yeast withstands 125° C.
Lower plants and animals	Spirogyra killed in water at 45°–50°.[a] Many algæ live in the Carlsbad thermal springs, temperature 53° C. Some forms of algæ have been found in thermal springs having a temperature of 85° C.
	Salt-water amœbas are killed at 35° C.; fresh-water amœbas at 40°–45° C.
	Strong light is harmful.
Zymase (yeast)	Optimum temperature, 25° C.; loses activity at 0° C. and is destroyed at 53° C.
Maltase or glucase	Yeast maltase destroyed at 55° C. Maize maltase acts best at 35° C.
Invertase (from yeast)	Quickly destroyed when moist at 70° C. and at 50° C. when heated a a long time. Optimum temperature, 31° C.; according to Kjeldahl, 52°–56° C.
Diastase (of malt)	Moist heat kills at 75° C, after short exposure. Dry diastase stands 100° C. and over. Optimum temperature, 50°–55° C.
	Sunlight kills it.
Emulsin	Optimum temperature, 45°–50° C.; destroyed at 70° C. In dry state stands 100° C. for hours.
Myrosin	Inactive at 0° C.; thermal death-point, 85° C.
Pepsin (from the stomach)	In 0.2 per cent hydrochloric acid, optimum temperature, 35°–40° C. Death-temperature, 56°–60° C. Dry, stands 160° C. for a short time.
Trypsin (from pancreas)	Optimum temperature, 40° C.; death-temperature, 69°–70° C. In dry state can be heated to 160° C. without harm.
Papain (vegetable trypsin)	Optimum temperature, 40° C. Killed at 75° C. Dry, stands 100° C.
Rennin	Optimum temperature, 40° C. Higher than 70° C. kills quickly.
Catalase (Loew)	Death-temperature, 72°–75° C.
Laccase (Bertrand)	Optimum temperature, 20° C. Destroyed at 60°–63° C. (Yoshida). Destroyed at 70° .C. (Bertrand).

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[a] The water plant Hottonia shows a maximum assimilation of carbon dioxide at 31° C. This is only one fourth as strong at 50° C., and at 56° C. it ceases altogether.

PART II.—(2) CHANGES IN THE COMPOSITION OF MILK PRODUCED BY THE MILK ENZYMES.

According to Marfan (1), milk is not an inactive fluid, but possesses properties which are more or less characteristic of living tissues. For example, it gives Bordet's reaction, namely, that when the milk of one species of animal is repeatedly injected into the blood of an animal of different species the blood serum of the animal so treated gradually acquires the power of coagulating milk in much the same way as rennin. (See P. T. Müller (2) "Vergleichende Studien Ueber die Gerinnung des Kaseins Durch Lab und Lactoserum.") Human milk is said by Moro (3) to have the power of coagulating hydrocele fluid, whereas cow's milk does not give this reaction.

As already indicated on page 313, milk contains a number of soluble ferments, such as diastase (amylase), galactase, lipase, lactokinase, peroxidase, reductase, and catalase.

In the present state of our knowledge we know very little of the actual functions of the milk ferments. According to Marfan (1), it is probable that the milk ferments act as stimulants and regulators of nutrition and that they are identical in function with the enzymes elaborated by the various tissues and are intended to compensate for the deficiency of the internal secretions of the new-born. According to this author the presence of specific ferments in the milk of a particular animal species probably explains the value of natural over artificial milk feeding.

Engel (4), in discussing Moro's work on the influence of the milk ferments on nutrition, arrives at the conclusion that while these ferments are apparently characteristic for the milk of any particular animal species, we can not yet come to any definite conclusions respecting their influence on animal nutrition. He calls attention to the fact that Moro's curves showing the increase in weight of two sucklings both fed by the bottle, one on fresh and the other on boiled human milk, showed but little differences. Both curves showed that both children thrived less well during the second period. Concerning the function of the milk ferments see also Moro (5).

On the other hand certain of these ferments bring about changes in the composition of milk which require some consideration in this connection. The following are the principal facts of interest concerning the soluble ferments found in milk and the changes which they effect in the composition thereof, except for what has been given already on this subject.

Diastase (amylase) of milk.—In 1883 Béchamp (6) isolated from human milk an enzyme which liquefies starch and converts it into sugar as readily as diastase. According to this author this ferment was obtained from successive portions of milk as soon as drawn from the teat, and hence is a product of the milk gland itself, and not formed by the action of milk stagnated in the gland. Attempts to isolate this ferment from cow's milk by Moro (5) and by Van der Velde and Landtsheer (7) have not proven successful. That a diastatic ferment does not occur in cow's milk has also been confirmed by Kastle. At present nothing definite is known regarding the function of this enzyme in human milk, and so far as we know it is not responsible for any alteration in the composition of any constituent of the milk itself.

Galactase.—This proteolytic ferment was first recognized by Babcock and Russell (8) in 1897, and has been found by these observers in the milk of the cow, woman, sheep, goat, pig, horse, and half-breed buffalo. By the methods ordinarily employed in the preparation of enzymes, these authors succeeded in preparing from the fresh centrifuge slime of milk that had been kept continuously in contact with chemical antiseptics aqueous extracts possessing proteolytic properties to a marked degree. These extracts were also observed to have the power of curdling fresh milk, similarly to rennin, and also of rapidly decomposing hydrogen peroxide. Galactase has been found to be similar to trypsin in its action on proteids, converting them into proteoses and peptones and finally into amino-acids. Like trypsin it has been found to be most active in solutions that are slightly alkaline to litmus, and like all ferments it is easily destroyed by heat.

Some idea of the changes produced in milk by the action of this enzyme may be formed from the results of analyses made by Babcock and Russell of milks that were allowed to stand for various intervals of time in the presence of an antiseptic to prevent the growth of bacteria. These results are given in the following table:

Description of milk	Per cent of proteids in soluble form.
Average of fresh whole milks analyzed	21.07
Average of whole milks, 20-25 days old	38.27
Average of centrifugal skim milks (fresh)	25.26
Average of centrifugal skim milks, 8-12 months old.	73.30
Maximum found in skim milk	91.18

The proof of the enzymic nature of these changes is shown by the stability of milk heated to a sufficiently high temperature to destroy such ferments, and by the fact that fresh milks when preserved with powerful antiseptics, such as mercuric chloride, formalin, etc., undergo no change even though they be kept for indefinite periods of time.

These observations on galactase have been fully confirmed by Von Freudenreich (9) and other investigators (10). Wender (11) has shown, however, that galactase as ordinarily prepared from separator slime, according to Babcock and Russell's method, in reality consists of at least three distinct enzymes, viz, gallactase proper, peroxidase, and catalase. Ordinarily galactase by itself acts too slowly to cause any material change in the proteids of milk in the short intervals which usually elapse between the withdrawal of the milk from the animal and its consumption as food. It is claimed by Babcock and Russell, however, that this enzyme probably assists in those changes which ordinarily take place in the ripening of cheese. It is also claimed by Snyder (12) that when milk is used in a mixed diet the proteids have been found to be from 4 to 5 per cent more digestible than when milk is omitted from the diet. This increased digestibility he claims is due to the milk enzymes. In this connection, it is of interest to note that Hougardy (13) has recently shown that cow's milk contains a ferment or a kinase similar to enterokinase. The author proposes to call this ferment lactokinase. This lactokinase has been found to accelerate the digestion of proteids by pancreatic juice and loses its power to facilitate this change at 73° to 75° C.

Lipase.—Marfan and Gillet (14) found a lipase in milk capable of hydrolyzing monobutyrin. Human milk exhibits this property to a higher degree than cow's milk. The former was found to have a lipolytic activity of 20–30 on Hanriot's scale, while cow's milk shows an activity of only 6–8. Gillet (15) has shown that the milk of different animals contains the lipolytic ferment. This ferment withstands cold, but is destroyed by heating to 65° C. It is nondialyzable and is held back by the porcelain filter. It probably hydrolyzes the higher fats of milk at least to some extent and may possibly account for a small part of the acidity of sour milk.

In this connection Rogers (16) has observed that this ferment is present in butter and on standing increases its acidity.

The so-called "salol-splitting ferment."—Nobécourt and Merklen (17) observed that human and ass's milks have the power of hydrolyzing salol (phenyl salicylate). For a time this hydrolysis was believed to be accomplished by an enzyme, to which the name of "the salol-splitting enzyme" was given. It was afterwards shown, however, by Desmoulières (18) and also by Miele and Willem (19) that no such ferment exists in milk and that this decomposition of salol is in reality a saponification brought about by the alkali present in certain milks, and that only those milks having an alkaline reaction are capable of effecting this decomposition, so that this probably disposes of this subject.

The oxidizing ferments of milk.—Milk contains no true oxidases or oxidizing ferments proper. It does decompose hydrogen peroxide, however, and in the presence of small amounts of hydrogen peroxide or ozonized oil of turpentine it has the power of effecting the oxidation of a considerable number of easily oxidizable substances. In other words, milk contains catalase and peroxidase. These have been referred to in the literature by these names; and also more or less indiscriminately by certain writers as the oxidizing ferments of milk or superoxidases, and also by some as the indirect oxidases.

Catalase.—From what is known of the wide distribution of the catalases among living things and plant and animal secretions it seems probable, although it can not be said to be known absolutely at present, that the fresh milk of all animals has the power of decomposing hydrogen peroxide more or less easily. Jolles (20) has pointed out that human milk decomposes five or six times as much hydrogen peroxide in the same length of time and under the same conditions as cow's milk. This author is inclined to attach considerable importance to this reaction, and recently Von der Velden (21) also lays emphasis on the fact that the presence of catalase in human milk serves to distinguish it from cow's milk. On the other hand, the fact that cow's milk can decompose hydrogen peroxide is attested by many observers, some of whom, among them Amberg (22), have called attention to the gradual disappearance of hydrogen peroxide in cow's milk on standing, and others, van Itallie (23) among them, to the fact that cow's milk loses its power to decompose hydrogen peroxide on heating to 63° C. Faitelowitz (24) has shown that the catalase of milk is associated with the fat globules. This has been confirmed by Reiss (25), who further points out that the catalase of milk is insoluble in the presence of colloids.

In the present state of our knowledge we know very little concerning the function of catalase in living tissue and active secretions. The view has been advanced by Loew (26), who has made extensive studies in this field, that the function of catalase is to destroy any hydrogen peroxide that may have been formed during the respiratory processes in the living cell, thereby preventing the accumulation of this and other peroxides, all of which are more or less toxic in their effects, thus affording protection against a toxic product of respiration. The question whether hydrogen peroxide is formed in the respiratory process in plants or animals is a much-mooted question, and there has been considerable difference of opinion among chemists as to whether hydrogen peroxide ever occurs in animal or vegetable tissues. One thing is certain, however, and that is, whether hydrogen peroxide occurs therein or not other complex peroxides do occur, especially in plant tissues and exudations (see Bach's (27) investigations), and quite recently in an investigation of remarkable interest and far-reaching importance, Usher and Priestley (28) have confirmed Erlenmeyer's (29) theory of the origin of carbohydrates in green plants, which supposes these substances to originate from formic aldehyde, which in turn results from the reaction of carbon dioxide with water, as indicated in the following equations:

$$(1)\ CO_2 + 2H_2O = \overset{\text{formic acid}}{H_2CO_2} + H_2O_2$$

and

$$(2)\ H_2CO_2 + H_2O = \overset{\text{formic aldehyde}}{H_2CO} + H_2O_2.$$

In this reaction hydrogen peroxide is formed, and of course if allowed to accumulate in the cell would soon put a stop to all of its vital activities. According to Loew, however, the accumulation of hydrogen peroxide would be prevented by its decomposition, practically as fast as formed, by the catalase.

Quite recently Usher and Priestley (30) have succeeded in demonstrating the decomposition of carbon dioxide by means of chlorophyll within and outside of the plant and in proving the presence of formic acid, formic aldehyde, and hydrogen peroxide among the products of the decomposition. As already stated this work is of unusual interest, and if confirmed by subsequent investigations will go a long way toward bringing about an understanding of this important biochemical process and will enable us to understand better the function of the catalases in general. Of course if hydrogen peroxide or similar peroxides are present in milk they must of necessity have a different origin from the peroxides occurring in the cells of chlorophyllous plants. It is readily conceivable, however, that such peroxides might originate in animal tissues and secretions in other ways, and if, in the one case, it is established that the function of catalase is to destroy hydrogen peroxide and thereby prevent its accumulation in the cell, it will probably turn out that it has a similar function in milk or in whatsoever associations it is found.

It is held by some that the catalases are not oxygen carriers, and in this connection Lesser (31) has shown that the decomposition of hydrogen peroxide by catalase does not lead to the oxidation of fat or carbohydrates. According to this author catalase is to be regarded as a substance capable of taking up oxygen and of giving it up again under certain circumstances.

Peroxidases.—The idea that milk contains true oxidizing ferments or oxidases proper probably originated from the fact that in the earlier work on this subject old tinctures of guaiacum were employed in making the tests. As is well known, old tinctures of guaiacum frequently exhibit reactions which are not shown by the perfectly fresh tinctures (32). This has been accounted for on the supposition that on standing exposed to air and light substances of the nature of peroxides are gradually formed in tinctures of guaiacum and that these substances react with the unchanged guaiacum in the presence of a peroxidase or suitable oxygen carrier, giving rise to the formation of guaiacum blue. On the other hand, there is abundant evidence at hand to show that milk contains substances capable of inducing the oxidation of guaiacum and other readily oxidizable substances by means of hydrogen peroxide or ozonized oil of turpentine. These substances are destroyed by boiling and are known as the peroxidases. A great many reagents have been proposed for the detection and approximate estimation of the peroxidases in milk, with the view, primarily, of distinguishing between fresh or raw and heated (pasteurized) or boiled milk. Among these may be mentioned guaiacum (33), potassium iodide, and starch (34), paraphenylene-diamine (35), ortol (36), paradiethyl-paraphenylene-diamine (37), ursol (38), guaiacol (39), amidol (Leffmann) (35), phenolphthalin (40), benzidine (41), etc. These reagents are used in connection with small quantities of hydrogen peroxide or some peroxide compound such as the persulphates, perborates, or ozonized oil of turpentine, and with fresh unheated milk they all give characteristic changes of color which are not shown by milks which have been heated to 80° C. or higher.

Whether the peroxidases of milk give rise to any changes in the composition of the milk can at present only be conjectured. It may be of course that they gradually effect the oxidation of reducing substances in the milk. According to some authors they gradually disappear when the milk turns sour. It has been our experience, however, that they pass practically unchanged into the whey when milk curdles as the result of the lactic acid fermentation. In the present state of our knowledge the various tests which have been proposed for the peroxidases of milk are chiefly useful in enabling us to form an idea of the condition of the milk, whether it has been heated beyond certain temperatures or not, although according to Gillet (15) even normal fresh milks vary in the amounts of peroxidases which they contain, and this has also been our own experience with this reaction.

Reductases.—According to Seligmann (42) raw milk possesses reducing properties; for example, it reduces Schardinger's (43) reagent, which consists of a solution of methylene blue containing small amounts of formaldehyde. By some authors these reducing substances have been regarded as ferments, reductases, by others as due to bacteria, and by still others they have been looked upon as identical with catalase, the ferment in milk which decomposes hydrogen peroxide. By the use of a weak alcoholic solution of methlyene blue, Smidt (44) claims to have been able to distinguish between the reduction brought about by bacteria and that caused by ferments. This author has shown that the reductases of milk are different from the catalase and superoxidase of milk and separable from these, the latter being soluble in water and salt solution, the former not. In recent communications Seligmann (45) points out that the reductases and peroxidases of cow's milk are not indentical. According to this author all processes of reduction occurring in fresh and sour milk are due to the action of bacteria and not to unorganized ferments.

Cathcart (46) has also made a study of the reduction of Schardinger's reagent by fresh milk. According to this author the reduction of the coloring matter is due to the presence of a catalase which is readily destroyed by heat. Our knowledge, therefore, of the reductases of milk is at present very limited, and we are not as yet in a position to say whether they are responsible for any of the changes ordinarily occurring in milk.

Part II.—(3) Changes in Milk Brought About by the Action of the Digestive Ferments—The Rennin Coagulation of Milk.

The composition of milk is profoundly altered during the process of digestion through the action of the digestive ferments. In the stomach and intestine the fat is hydrolysed by lipase, giving rise to fatty acids and glycerin, the milk sugar is converted into glucose and galactose by lactase, and the proteids into simpler and more disfusible nitrogen compounds by the proteolytic ferments. Chief among these proteids is caseinogen, which, according to Lehmann and Hempel (1), has the following composition:

	Per cent.
Carbon	54.
Hydrogen	7.04
Nitrogen	15.6
Sulphur	.771
Phosphorus	.847

The following, according to Mann (2), are the principal dissociation products which have been isolated from caseinogen by hydrolytic cleavage:

	Per cent.
Glycocoll	0
Alanin	.9
Leucin	10.5
Phenylalanin	3.2
Alpha pyrrolidin carboxylic acid	3.2
Glutaminic acid	10.7
Aspartic acid	1.2
Cystin	.065
Serin	.43
Oxy-alpha-pyrrolidin carboxylic acid	.25
Tyrosin	4.5
Lysin	5.8

	Per cent.
Histidin	2.6
Arginin	4.84
Tryptophane	1.5
Ammonia	1.8
Cystein	0
Amino valerianic acid	1.
Glucosamin	0
Diamino-trioxy dodecanoic acid	.75

According to this author the absence of glycocoll and the carbohydrate radical and the relatively high tyrosin and tryptophane content of caseinogen render it especially readily digestible. It seems also to be the only native albumin which is attacked by erepsin (see Cohnheim (3)), and on account of its ease of hydrolysis it probably plays a special port in metabolism. In this connection Tunnicliffe (4) has shown that when total digestibility is considered human milk is much more digestible than any of its substitutes.

Among the various changes brought about in the composition of milk through the action of the digestive ferments the most typical and characteristic is the rennin coagulation. Exclusive of the mineral matter the caseinogen is the only constituent of the milk involved in this change.

It has long been known that fresh milk coagulates in the stomach of higher animals, and that an aqueous extract of the inner lining of the stomach of the calf, when added to fresh milk, causes it to curdle, whereby it clots or sets in the form of a solid curd. Since early times this fact has been turned to practical account in the making of cheese. The earlier explanations of the rennin coagulation of milk were based on an observation by Fremy (5) to the effect that rennet, or the mucous lining of the calf's stomach, has the power of converting milk sugar into lactic acid. According to Liebig (6), therefore, rennin curdles milk for the reason that it acts upon the milk sugar, converting it into lactic acid. The latter then neutralizes the alkali of the milk which holds the caseinogen in solution, thereby precipitating this substance as the curd.

Soxhlet (7) also saw in the curdling of milk by rennin an analogy to the coagulation of milk by acids. According to him the former process took place much more rapidly than the latter. He held with Liebig that the rennin converted the sugar of milk into lactic acid and that this in turn converted the alkaline phosphate existing in the milk into an acid phosphate, which in turn precipitatel the casein. Hallier (8) explained the rennin coagulation of milk as due to the presence of micro-organisms in the stomach of the calf. The most important of the earlier observations on the curdling of milk by rennin was that made by Heintz (9), who showed that contrary to previous teachings on the subject the acqueous extract of the mucosa of the calf's stomach has the power of curdling milk in both acid and alkaline solutions. To Hammarsten (10) and Schmidt (11), however, belong the credit of first showing that the rennin curdling of milk is accomplished by means of a soluble ferment to which they gave the name of "labferment" or "chymosin." This is the ferment which in English is called rennin, formerly rennet. Hammarsten succeeded in showing: first, that the curdling of the milk by rennin is independent of the action of lactic acid; second, that the caseinogen (casein) of milk is not in true solution in milk but in colloidal suspension (gequollenen Zustande); third, that without the presence of a sufficient quantity of calcium phosphate rennin coagulation will not take place; fourth, that the caseinogen is so modified through the action of the rennin that in the presence of a certain quantity of a lime salt it can no longer remain in solution, but is precipitated as casein (Käse) or paracasein calcium phosphate; fifth, that as the result of the action of rennin, caseinogen (casein) is split into at least two new proteids, casein (der Käse) and wheyproteid (Molkeneiweiss). The former contains a relatively small quantity of calcium salts and is insoluble, the latter contains a larger proportion of calcium salts and is easily soluble. Finally Hammarsten held it to be highly probable that the rennin coagulation of milk is analogous in many respects to the coagulation of fresh milk by heat, which occurs at 130° to 150° C., and that in this regard the action of rennin is similar to other fermentations. According to Hammarsten, therefore, the rennin coagulation of milk resolves itself into two distinct phases: (1) the conversion of caseinogen[a] into paracasein in the presence of calcium salts, (2) the precipitation of paracasein from its solutions through the action of calcium salts. It will be observed that the second phase of the coagulation is independent of the action of rennin.

These earlier researches by Hammarsten on the rennin coagulation of milk have been the point of departure for the greater number of subsequent investigations in this field, and his conclusions respecting this process have been the subject of a great deal of discussion.

During recent years the rennin coagulation of milk has been studied by many observers. Among these may be mentioned Duclaux, Courant, Lörcher, Fuld, Laqueur, Loevenhart, and others. As the result of his studies on the rennin coagulation of milk, Loevenhart (14) recognized essentially three distinct phases of the process: (1) Transformation of caseinogen into paracasein; (2) alteration or rearrangement of the mineral constituents of the milk, whereby the calcium salts become available for the coagulation; (3) precipitation of the paracasein by calcium salts. He has shown that the conversion of caseinogen into paracasein proceeds somewhat more rapidly than the rendering available of the calcium salts. According to this author the first two phases of the process are accomplished by the action of rennin, whereas the third phase, namely, the precipitation of the paracasein, is entirely independent of the action of the ferment.

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[a] The name caseinogen is employed throughout this communication on the rennin coagulation of milk in the sense in which it was first used by Halliburton (12), namely, as signifying the proteid of milk, which, through the action of rennin in the presence of certain calcium salts, is transformed into the casein (paracasein) of the curd. The term paracasein was first introduced into the science by Schulze and Rose (13) and is used in the sense employed by Hammarsten.

He also arrived at the conclusion from his study of the influence of salts on the coagulation of decalcified milk that the facts observed seemed to favor the theory that the curdling of milk depends in great part, though not entirely, on the rearrangement or rendering available of its mineral constituents. He succeeded in showing that fresh milk can not precipitate paracasein solutions nor can it prevent their precipitation by calcium chloride. Hence it would seem that the calcium salts of fresh milk are in some way altered through the action of rennin, thereby becoming capable of precipitating paracasein. He concludes therefore that in the rennin coagulation of milk the rennin has the power in some way to render available the calcium salts (die calcium Salze frei zu machen), since without this change no coagulation is possible. Similarly Briot (15) maintains that rennin acts less on the caseinogen than on the calcium phosphate of milk. While this extreme view is probably incorrect it is certain that the majority of chemists are agreed regarding the necessity of calcium salts for the rennin coagulation of milk. That such is the case is evident not only from the earlier investigations of Hammarsten but also from later and more exact observations by Arthus and Pages (16), Courant (17), Ringer (18), Loevenhart (14), Edmunds (19), Benjamin (20), Söldner (21), Laqueur (22), and others.

It has also been established by the work of Courant (17) that the reaction of milk is not altered during the rennin coagulation.

The question still remains to be considered, How does the rennin act on the caseinogen and in what way is the latter altered through the action of the ferment? These questions have been exhaustively considered by Laqueur (23), who has arrived at the conclusion that from the slight differences between caseinogen and paracasein thus far made out it is impossible to arrive at an unequivocal explanation of the coagulation of milk by rennin. This author is inclined to believe, however, that Hammarsten's original explanation of the process is perhaps, all things considered, the best we have. According to this explanation the rennin acts by splitting the caseinogen into a larger molecule, paracasein, and a smaller molecule, the whey proteid (molkeneiweiss), also called hemicaseinogen albumose (Arthus and Pages). Fuld (24) has recently suggested the name whey-albumose for the soluble proteid produced in the rennin coagulation of milk. This view also derives support from the more recent researches on the subject by P. T. Müller (25), who found whey-proteid in the milk serum only after rennin coagulation and not after the milk had been coagulated by acids or lactoserum. Similarly, unpublished analyses by W. Laqueur (23) and an experiment by Rotondi (26) also indicate the splitting off of a soluble nitrogenous compound from caseinogen during the rennin coagulation of milk. On the other hand, according to Duclaux (27), who analyzed the filtrates obtained by filtering fresh milk and milk coagulated by rennin through a porcelain filter, the soluble nitrogen in the whey is not increased after rennin coagulation, nor is the composition of the whey altered in any way. That such is the case may be seen from the following results of his analyses of milk serum before and after coagulation by rennin:

	Experiment I.		Experiment II.	
	Normal milk.	Milk coagulated by rennin.	Normal milk.	Milk coagulated by rennin.
Lactose	5.53	5.53	5.37	5.64
Soluble albumin	.55	.57	.37	.36
Mineral matter	.54	.52	.56	.40

Arrhenius (28) is of the opinion, however, that Duclaux's experiments on this point are not convincing, since the whey proteid might have been retained by the porcelain filter, especially in the presence of the gelatinous paracasein.

From his researches on the laws governing rennin coagulation Fuld (29) also arrived at the conclusion that the transformation of caseinogen into paracasein is only a molecular rearrangement, partaking of the nature of a monomolecular process. According to this author the rennin coagulation of milk is only a special case of the well-known phenomenon of the reciprocal suspension and precipitation of colloidal substances. Loevenhart (11) has also reached the conclusion that it is probable that caseinogen and paracasein are chemically the same substance, and that the observed differences existing between them depend upon the fact that paracasein exhibits a higher degree of association than caseinogen. In other words, paracasein consists of larger molecular aggregates than caseinogen, otherwise they are identical. These views are shared by other observers, among them Van Slyke and Hart (30). On the other hand, Laqueur (23), from a consideration of these facts and the conduct of other colloidal substances, comes to exactly the opposite conclusion, viz. that caseinogen is probably a higher colloid than paracasein.

According to Laqueur (23), the idea that paracasein is a more complex substance than caseinogen has also received support from Danilewski's observation that rennin gives rise to a precipitate in solutions of albumoses. According to the Russian investigators, by whom this reaction has been extensively studied, this precipitate possesses the properties of a higher proteid. They therefore see in this change the synthesis of a complex substance having the nature of naturally occurring proteids from the products of assimilation, and find a ready explanation for the widespread occurrence of rennin in the tissues of animals and plants which are in no wise concerned with the digestion of milk. On the other hand, Laqueur (23) is inclined to question the validity of these conclusions. According to this author the weightiest objection that can be urged against these views is that at present we have no exact means of knowing whether the reaction resulting in the formation of these plastein substances, as they have been called, is really the result of rennin action and whether we have a right to ascribe to these changes the same cause as that which brings about the conversion of caseinogen into paracasein in the coagulation of milk. We know that as yet we have no means of operating with the pure ferment, but that in a solution of ferments the ferment itself often composes only a small part of the mixture, and in this connection it has been found that whereas one part by weight of the ferment solution is required to convert 18 grammes of albumose into plastein the same quantity of the ferment solution will convert from 10,000 to 100,000 grammes of caseinogen into paracasein. Hence, according to Laqueur (23), it would seem to be a far-fetched conclusion to ascribe these two changes to the same ferment. It is therefore a mistake, according to this author, to assign to a ferment so widely distributed in plants and animals as that causing the plastein reaction a function absolutely identical with that of the ferment contained in the stomach of the calf, the latter producing the typical rennin coagulation of milk, until it has been definitely established that the ferment from plants, etc., also acts on caseinogen in two stages, in one of which calcium salts are required, and that the paracasein produced in the two processes is the same in each. Other chemists are also of the opinion that the plastein reaction is in reality due to pepsin and not to rennin at all.

As a matter of fact the chemical and physical differences between caseinogen and paracasein are apparently so slight and in the present state of our knowledge so imperfectly understood that it is impossible to decide between all of these conflicting views on the rennin coagulation of milk. Laqueur (23) takes the view, however, that the hypothesis that rennin causes a coagulative splitting of caseinogen rests at least upon some foundation of fact, whereas the view that rennin exerts a synthetic action rests at present upon very deceptive analogies and teleological evidence.

The more one studies the extensive literature of the rennin coagulation the more one is disposed to agree with Laqueur that in the present state of our knowledge it is impossible to arrive at an unequivocal explanation of this complicated process. It seems, however, to have been reasonably well established—

(1) That, exclusive of phosphates of calcium and other soluble salts of calcium, caseinogen is the only substance in milk involved in the rennin coagulation.

(2) That in the rennin coagulation of milk no change of reaction occurs; that is, no production of base or acid. In this connection it has been pointed out by Herwerden (31) that hydrogen ions are not necessary for the rennin coagulation of milk or of solution of caseinogen containing calcium.

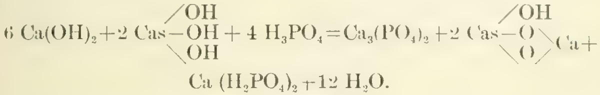
(3) That in the rennin coagulation of milk two active agents are concerned, a soluble ferment, rennin, and calcium ions; that is, soluble calcium salts.

According to Hammarsten the caseinogen is resolved by rennin into paracasein and whey proteid. Through the action of calcium ions (soluble calcium salts), the former is then precipitated as the curd (Käse), the latter remaining in solution. According to Fuld and others the change of caseinogen into paracasein is a molecular rearrangement. According to Courant the dicalcium caseinogenate is so altered by rennin that by contact with soluble calcium salts a precipitate (the curd) is produced. According to Lovenhart the rennin renders the calcium salts of milk available for the coagulation of paracasein, which latter is formed from caseinogen also by the action of rennin, and which, according to this author, differs from caseinogen only in that it is composed of larger molecular aggregates. It will be observed that these several views regarding the precise mode of action of the rennin differ in some particulars. These differences can only be reconciled by further investigation.

It has been shown by Söldner (32), Osborne (33), Courant (17), and others that caseinogen is an acid. According to Courant it forms three kinds of salts, namely, mono-, di-, and tri-caseinogenates. It also seems probable from Lehmann's (1) investigations that the caseinogen exists in fresh milk in the form of a complex calcium salt containing calcium phosphate. According to this author the composition of this compound agrees reasonably well with the formula

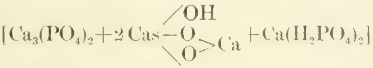
$$Ca_3\,(PO_4)_2\ Ca.\ \text{caseinogen.}$$

According to Courant (17) a solution can be obtained showing essentially the same alkalinity to lacmoid and the same acidity to phenolphthalein and conducting itself to rennin in the same manner, as fresh milk, by bringing together lime water, caseinogen, and phosphoric acid in the quantities indicated in the following equation:



$$6\,Ca(OH)_2 + 2\,Cas\begin{array}{l}/OH\\-OH\\\backslash OH\end{array} + 4\,H_3PO_4 = Ca_3(PO_4)_2 + 2\,Cas\begin{array}{l}/OH\\-O\\\backslash O\end{array}\!\!>Ca + Ca\,(H_2PO_4)_2 + 12\,H_2O.$$

In other words, the mixture or compound



$$\left[Ca_3(PO_4)_2 + 2\,Cas\begin{array}{l}/OH\\-O\\\backslash O\end{array}\!\!>Ca + Ca(H_2PO_4)_2\right]$$

closely approximates the condition of the caseinogen and phosphates as these substances exist in fresh milk. Whether these several substances are merely intimately mixed together or whether they form some loose chemical combination similar to a complex double salt can not be definitely determined at present. According to Courant, however, all of these substances are necessary to the rennin coagulation, namely, dicalcium caseinogenate, soluble calcium salts, represented in the equation by monocalcium phosphate, and also tricalcium phosphate. According to this author only the dicalcium caseinogenate is altered in the rennin coagulation of milk, this being converted into paracasein. According to him the rôle of the soluble salts of the alkaline earths (calcium) in this process is simply to diminish the solubility both of the caseinogen itself and the paracasein. This last notion is in harmony with certain observations by Ringer (18), who found that even fresh milk is coagulated by warming with small amounts of calcium salts. According to this author three drops of a solution of calcium chloride are sufficient to curdle ten cubic centimeters of fresh milk at 70° to 75° C. He further observed that while a very slight acidity seems to favor the coagulation by calcium salts it is by no means essential to the process, since it can be brought about even in faintly alkaline milk.

Finally, in the presence of soluble calcium salts the paracasein resulting from the action of rennin is precipitated in the form of an insoluble calcium salt containing calcium phosphate, either in loose chemical combination or as an intimate mixture. In this connection it has been shown by Harris (34) that in the curdling of milk by rennin 13 per cent more calcium phosphate is used up than in the acid coagulation of milk.

In this connection Courant's (17) views regarding the composition of milk and the manner in which the caseinogen is held therein afford the most satisfactory explanation of the conduct of fresh milk toward chemical indicators. To review this subject briefly, Courant has found that cow's milk and caseinogen solutions, such as that whose composition is given in the above equation, react alkaline to lacmoid and acid to phenolphthalein. The acidity of fresh cow's milk proved to be slightly less than that of the caseinogen solutions; the alkalinity of milk, on the other hand, was nearly twice that of the caseinogen solutions. He reaches the conclusion that one half of the acidity toward phenolphthalein as shown by cow's milk and his caseinogen solutions is due to the acidity of dicalcium caseinogenate, and the other half to monophosphates. In this calculation he purposely neglects the slight acidity of milk due to the free carbonic acid which it contains. The alkaline reaction toward lacmoid depends in the case of the caseinogen solutions partly on the dicalcium caseinogenate, which, like the salts of other weak acids, is readily hydrolyzable, yielding a certain amount of free base, namely, calcium hydroxide, and partly on the insoluble phosphates. In the case of milk the alkalinity toward lacmoid depends on these two factors and also on the presence of diphosphates. The greater alkalinity of cow's milk depends partly on the larger quantity of insoluble phosphates present, but principally on the presence of diphosphates. As has been repeatedly shown, human milk is more alkaline than cow's milk. According to Courant, however, it, like cow's milk, is also acid to phenolphthalein and alkaline to lacmoid. In the case of cow's milk he found the ratio of alkalinity to acidity to be $\frac{4.1}{1.95}=2.1$, and in the case of woman's milk $\frac{1.08}{0.36}=3$. According to this author the relatively slight acidity of woman's milk is due to the small quantity of caseinogen which it contains and also in all probability to the fact that it contains its caseinogen in the form of tricalcium caseinogenate. To return for a moment to the subject of the rennin coagulation of milk, it would seem that certain aspects of this change exhibit an analogy to the action of a toxin. It has been shown, for example, by Hammarsten and Roden (35) that normal horse serum contains a substance capable of inhibiting the action of rennin. In other words, it contains an antirennin. Similarly, by repeated injection of small amounts of rennin into the blood of animals, Morgenroth (36) obtained an antirennin. According to Fuld and Spiro (37) the antirennin of normal horse serum prevents the coagulation of milk by binding the calcium ions. Arrhenius is therefore of the opinion that in these reactions rennin coresponds to the toxophorous group, the calcium ions to the haptophorous group of a toxin, and the antirennin to an antitoxin.

Many additional facts concerning the rennin ferment are known. Like other ferments, it is affected by heat, and the rate of the rennin coagulation is determined both by the quantity of rennin acting and by the temperature. It has been shown that the ferment can withstand a temperature of —180° C. without injury. At temperatures higher than 41° C. the ferment gradually loses its activity, and exposure to a temperature of 50° to 60° C. for a considerable time has been found to be more harmful than a short exposure to a higher temperature. The effect of temperature is also determined by conditions surrounding the ferment, whether it is moist or dry, and also by the reaction of the solution containing the ferment. In the dry state it can withstand a temperature of 100° to 140° C. Its destruction by heat has been found to follow the law for a monomolecular reaction.

The influence of temperature on the rennin coagulation has been studied by Fuld (29). Some of his results are given in the following table:

Temperature ° (C.).	Time (sec.).	k observed.	k, calculated.
25.05	54	185	185
30	32	312	327
35	17	588	574
40	10.2	980	980
44	9	1,111	1,491
50	14.7	680	2,712

(The values of k, observed equal 10,000 divided by time.)

It will be observed that there is a good agreement between the observed and the calculated values up to 40° C. Above this temperature the observed values of k become smaller than the calculated values on account of the gradual destruction of rennin by heat.

In 1870 Segelke and Storch (38) showed that rennin coagulates milk in intervals which are inversely proportional to the concentration of the rennin solution. This conclusion has been confirmed by the later work of a number of observers. Thus Lörcher (39) obtained the following results from his measurements:

Quantity of rennin, in cc.	Time of coagulation, minutes.	Product.
0.01	....	....
.02	245	490
.03	155	465
.04	126.5	485
.05	92	460
.06	78	468
.07	69.25	485
.08	63	504
.09	56	504
.10	43	430
.20	24.5	490
.30	16	480
.40	12.5	500
.50	10	500
.60	8.75	525
.70	8.16	561
.80	7.5	600
.90	6.7	603
1.0	6.	600

Recently Madsen (40) has also investigated the effect of concentration of the rennin on the rennin coagulation, working at a temperature of 36.55° C. The following are the results of his measurements:

Time (minutes).	Rennin (grams).	Product.
4	0.08	0.32
6	.05	.30
9	.033	.30
11	.024	.26
12	.019	.23
14	.0175	.25
20	.013	.26
25	.01	.25
30	.007	.21
35	.007	.25
50	.005	.25
70	.004	.28
80	.0032	.26
100	.0028	.28
120	.0025	.30
180	.00185	.33
240	.0017	.41

The influence of various other factors, such as the reaction of the milk, the action of salts, the effect of ultraviolet rays, and the action of various organic substances on the rennin coagulation of milk has also been the subject of numerous investigations. It is ofttimes a difficult matter to determine whether these various influences are exercised toward the ferment itself or whether they react on the milk or participate only in the second phase of the rennin coagulation. The further consideration of such agents is beyond the scope of this communication. Before leaving the subject, however, it should be observed that Hillman (41) has studied the rennin coagulation of milk in its practical aspects. This author has found that the milk of fresh cows is better suited to the rennin coagulation than the milk of cows which are nearly dry. In his opinion this is probably to be explained by the diminution in the calcium content of milk during the period of lactation. He found, further, that the degree of acidity of milk in relation to the calcium content is an important factor. According to this author a high calcium content and high acidity prevail at the beginning of lactation and are usually accompanied by high total albumin and a high caseinogen content, all of which conduce to a large yield of paracasein. He also finds that the time of coagulation and the yield of paracasein are independent of one another; generally, however, a short coagulation time and a large yield of paracasein are associated. Strong dilution of the milk with water tends to diminish the yield of paracasein, whereas the addition of soluble calcium salts tends to increase it.

According to this author the action or rennin consists not only in the splitting of caseinogen into paracasein and whey proteid, but also in the conversion of other milk proteids into more soluble form. He seems to think that under favorable conditions paracasein may be formed from the albumin as well as from the caseinogen.

PART II.—(4a) CHEMICAL CHANGES IN MILK PRODUCED BY BACTERIA AND VARIOUS OTHER MICRO-ORGANISMS.

The more obvious changes in milk with which we are familiar are those that are brought about by bacteria and various other micro-organisms. Among these changes may be mentioned: The ordinary souring and curdling of milk, with the production of lactic acid as the chief product; the production in milk of various odoriferous or highly flavored substances, many of a somewhat disagreeable character, good examples being met with in the ripening of cream and cheese; the production of colored substances which impart to the milk unusual colors, such as the formation of blue milk; the formation of mucilaginous, or mucin like substances, which serve to impart to the milk a characteristic ropiness, known as ropy milk, and finally we must include under this head those bacterial changes in milk which result in the formation of poisonous substances, such as tyrotoxicon, toxins, etc.

The lactic acid fermentation of milk. The lactic acid fermentation is the commonest and best known of all the many bacterial changes that occur in milk. The fact that on standing at ordinary temperatures milk gradually turns sour and finally curdles has been known ever since milk was first used as a food by man. In early times the acid of milk was supposed to be acetic acid, the same as is present in vinegar, and as has already been pointed out this acid does, according to Béchamp (1), occur in even freshly drawn milk in small quantities. The substance really responsible for the souring of milk, however, viz, lactic acid, was first discovered in milk by Scheele in 1780. The new acid was also studied by Berzelius, and its composition definitely established through the work of Mitscherlich and Liebig in 1832. Its chemical constitution and its relation to other varieties of lactic acid, occurring in nature or the products of chemical synthesis, were first established by the labors of Strecker, Erlenmeyer, and Wislecenus.

In 1847, Blondeau (2) discovered micro-organisms in sour milk, but attached to these no particular significance so far as the souring of milk is concerned. It remained for Pasteur (3), in 1857, to definitely and conclusively show as one of the results of his classic investigations on fermentation that the souring of milk is really a kind of fermentation, which is accomplished by a peculiar kind of micro-organism, to which he gave the name of *levure lactique* (lactic yeast). His first communication on this subject was read to the Scientific Society of Lille, August, 1857, and afterwards to the French Academy in November, 1857. Since then our knowledge of the lactic acid fermentation has been considerably extended through the labors of Pasteur's students, and still later through the work of other bacteriologists and chemists. For example, Boutroux (4) in 1878, in continuing the investigations of Pasteur on the souring of milk, arrived at the conclusion that the lactic acid ferment and the mycoderma aceti, which is concerned in the transformation of alcohol into acetic acid in vinegar making, are identical, but that these vary in function, depending on their general environment and the composition of the liquid in which they grow. This communication also contains a description of the lactic ferment and an enumeration of its morphological characteristics, which are beyond the scope of the present communication. He observed that the organism grew best in a nutrient medium containing, besides albuminous matter, invert sugar or glucose. He also found that under these conditions the liquid can attain a maximum acidity of 1.5 per cent lactic acid. Larger amounts of acid than this checked the life and growth of the organism, and hence if it is desired to convert all of the sugar into lactic acid the acid must be neutralized with chalk or zinc carbonate as fast as formed. Under proper conditions the lactic acid organism employed by Boutroux produces lactic acid only.

The lactic acid fermentation of milk sugar was also investigated by Richet (5), who found that when milk is kept at 40° C. it becomes acid and coagulates and finally attains an acidity of 1.6 per cent, which amount it never exceeds. He made the further interesting observation that if gastric juice be added to milk the casein is coagulated and finally dissolved, and in less than twenty-four hours the milk contains a larger quantity of lactic acid than otherwise would have been produced in a week, and after four or five days as much as 4 per cent of lactic acid was formed. He observed that while neither a pure solution of lactose nor gastric juice will ferment, if the two be mixed fermentation takes place; and that the casein of milk after it has been dissolved by gastric juice also ferments, yielding lactic and butyric acids, besides other products of fermentation. On the other hand, the whey of milk obtained by coagulation with rennin never attains an acidity greater than 1.6 per cent of lactic acid, even after having been kept for six months. He found that the lactic acid fermentation is increased by exposing a large surface of the milk to the air. The activity of the ferment increases up to 44° C., remains constant between 44 and 52° C., and above 52° C. diminishes in activity as the temperature rises. Digestive juices and peptones were found to aid lactic fermentation, but leucine and glycocoll were found to have no effect upon the process.

The general trend of more recent investigations on the subject of lactic acid fermentation has been to show that the change of milk sugar into lactic acid takes place under the influence, either direct or indirect, of a whole series of micro-organisms, whose number has been considerably augmented by recent investigations in this field. Marpmann (6), for example, during the summer of 1885 investigated the micro-organisms of cow's milk in the neighborhood of Goettingen and detected five seemingly new and different species of organisms which more or less strongly induce the lactic acid fermentation in solutions of cane sugar and also in milk.

Leaving out of consideration the *levure lactique* of Pasteur, the first of these organisms whose morphological and biological characteristics seem to have been determined with sufficient accuracy is the Bacillus acidi lactici of Hueppe (7). It is now known that in addition to the Bacillus acidi lactici (Hueppe) the following organisms can bring about the lactic acid fermentation, viz. Bacillus aerogenes, Bacillus coli, Bacillus lactis acidi (Leichmann and others), Streptococcus lacticus (Kruse), Streptococcus pyogenes, Pneumonococcus A and Pneumonococcus B, Bacillus Delbruecki (Leichmann), Bacillus acidificans longissimus (Lafar), etc.

Beyerinck (8) has also made exhaustive studies of the lactic acid ferments employed in the arts. This author applies the name Lactobacillus Delbruecki to all species of the lactic-acid ferment which can be isolated by the gelatin-must method. These organisms, according to this author, however, are not the active agents of a good industrial ferment. On the other hand, from such a ferment he was able to isolate the Lastobacillus fermentans. This organism when cultivated under good conditions yields only lactic acid and no volatile acids. The minimum temperature of its activity he found to be 25° C., the optimum temperature 41°–42° C., and the maximum temperature 50° C. These observations furnish an interesting confirmation of the earlier work of Richet so far as the influence of temperature on the lactic fermentation is concerned. According to Beyerinck the lactic organisms studied by him can be mutually transformed into one another by cultivation.

Heinemann (9) has called attention to the similarity existing between Bacillus acidi lactici (Hueppe and others) and Bacillus (lactis) aerogenes (Escherich) and also to the similarity of Bacillus lactis acidi (Leichmann and others), Streptococcus lacticus (Kruse) and Streptococcus pyogenes, and in a more recent communication (10) on the kinds of lactic acid produced by lactic acid bacteria, to the similarity of Streptococcus lacticus with Streptococcus pyogenes and of Bacillus acidi lactici with Bacillus aerogenes. It has been observed by this author that in the ordinary souring of milk, lactic acid is produced chiefly by Streptococcus lacticus and Bacillus aerogenes, and further, that the former organism predominates in approximate proportion to the purity of the milk. Conn (11) also has shown that 95 to 100 per cent of all organisms in sour milk are of the Bacillus lactis acidi type (Streptococcus lacticus).

It is now known that lactose (sugar of milk) is not directly fermentable, but must first be converted into the simpler sugars glucose and galactose. It has been shown by a number of investigators that many yeasts and bacteria produce an enzyme which is capable of effecting this hydrolysis, and Hirschfeld (12) has shown that in the souring of milk the lactic acid bacteria accomplish the inversion of lactose, and that this change takes place most rapidly in the first thirteen to twenty-four hours after the introduction of the organisms into the milk, the relative amounts of lactose inverted being: First day, 0.16; second day, 0.23; third day, 0.29. Finally in this connection it has been shown by Buchner and Meisenheimer (13), and independently by Herzog (14), that an enzyme can be extracted from certain of the lactic-acid-forming bacteria, which in the absence of organisms is able to transform lactose and cane sugar into lactic acid. Buchner and Meisenheimer extracted the ferment from Bacillus Delbruecki (Leichmann), whereas in his experiments Herzog employed the Bacterium acidi lactici. On the other hand Beyerinck (8) is of the opinion that the production of lactic acid by the lactic acid bacteria is not a mere enzymic function, but a catabolic process.

The kinds of lactic acid produced in milk by the lactic acid bacteria.—As is well known, 4 isomers of lactic acid exist. Three of these are stereo-isomers of alpha-oxy-propionic acid or ethylidine lactic acid, the chemical structure of which is represented by the formula

$$CH_3.CHOH.COOH.$$

This compound, as indicated by the above formula, contains one asymmetric carbon atom, viz, the central one, and hence two optically active forms of lactic acid and one optically inactive form composed of equimolecular proportions of the two active varieties are possible and all three are known to exist. Aqueous solutions of one of these forms of lactic acid rotates the plane of polarization to the right, the second active variety rotates it to the left, and the third inactive form has no effect on the plane of polarization. Hence the first acid is called dextrolactic acid, and is designated as the d-lactic acid or simply d-acid; the second is called lævo-lactic acid and is designated as l-lactic acid or l-acid; and the third lactic acid of the above formula is called inactive or racemoid lactic acid and is designated (d + l) lactic acid or sometimes r-acid.

The fourth isomer of lactic acid has an altogether different chemical constitution from the other three forms of the acid and is known to chemists as beta-oxypropionic acid, hydracrylic acid, or ethylene lactic acid. It has the chemical structure represented by the formula

$$CH_2OH.CH_2.COOH.$$

This acid contains no asymetric carbon atom. It therefore exhibits no optical activity and only one form of it is known. This is not a product of the lactic acid fermentation, and hence does not further concern us in this connection. In the changes occurring in the fermentation of milk we are concerned with only the first 3 forms of the acid, viz, with the optical isomers of alpha-oxypropionic acid. Formerly it was generally accepted as pretty well established that the lactic acid produced in the souring of milk consisted mainly, if not entirely, of the r-acid. Indeed, ordinary lactic acid, viz, the r-acid, was frequently spoken of as fermentation "Gährungs" lactic acid. In 1895, however, it was shown by Günther and Thierfelder (15) that the lactic acid present in milk which has soured spontaneously does not always consist entirely of inactive lactic acid. They showed, in fact, that while the inactive acid was present in naturally sour milk there was often a preponderance of the dextro-rotatory acid. Further, the Bacillus lactis acidi (Streptococcus lacticus, Kruse) in pure lactose was found invariably to produce the d-acid.

In this connection Gadamer (16) has observed that commercial lactic acid is either inactive or dextro rotatory.

Quite recently Heinemann (10) has made an exhaustive study of the kinds of lactic acid produced in milk by the lactic acid bacteria. The following are his conclusions:

1. Milk naturally soured at room temperature contains chiefly d-acid. Milk soured at 37° C. contains chiefly r-acid with l-acid in excess if allowed to stand several days.

2. Streptococcus lacticus and Streptococcus pyogenes produce the same kind of lactic acid, i. e., d-acid. B. aerogenes from milk (B. acidi lactici) and the ordinary laboratory strain of B. (lactis) aerogenes (Escherich) produce the same kind of lactic acid, i. e., l-acid.

3. The lactic acid produced in naturally soured milk varies:

(*a*) According to the relative numbers of Streptococcus lacticus and B. aerogenes present, the higher the number of B. aerogenes the more l-acid is produced.

(*b*) According to the temperature at which the fermentation takes place, other conditions being equal, at 37° C. relatively more l-acid is formed than at room temperature.

(*c*) According to the length of time the fermentation has lasted, the longer the time the more l-acid is formed.

4. In "certified" milk, d-acid only was present at room temperature for nine days, while both d-acid and l-acid were present in milk of poorer quality after one to four days. At 37° C. l-acid was apparent after six days in "certified" milk and on the second day in other milk. It seems as if the purer the milk the longer the excess of d-acid persists.

5. Racemic lactic acid is the result of the formation of pure d-acid and pure l-acid by at least 2 different species of micro-organisms. Racemic lactic acid is not known to be the product of one species only.

6. Since it is known that B. aerogenes forms other acids besides lactic acid, often in appreciable amounts, while Streptococcus lacticus produces almost pure d-acid, the presence of d-acid may be taken as indicating desirable conditions for dairy work, because this shows the absence of the fermentation products of B. aerogenes; i. e., volatile acids, gas, and ethyl alcohol.

According to Claflin (17) in the lactic acid fermentation as carried out in the manufacture of the acid, in which process the acid produced is neutralized by chalk practically as fast as formed (or at least never allowed to exceed 0.02 to 0.5 per cent by weight of the solution), 98 per cent of the sugar is converted into lactic acid in three to six days through the action of a pure culture of Bacillus acidi lactici. On the other hand, in the ordinary souring of milk, in which case of course no pains are taken to neutralize the acid as it accumulates in the liquid, smaller amounts of lactic acid are formed and much less than the total quantity of lactose present is changed. Thus we have seen from the earlier observations of Boutroux and Richet that the lactic acid never exceeds 1.6 per cent by weight of the liquid undergoing fermentation, and according to recent observations by Blumenthal and Wolff (18), milk which has been kept four years may still contain 50 per cent of its original lactose unchanged.

It has also been found by Haacke (19) that the amount of lactic acid produced in the lactic fermentation never exceeds one-third of the amount of sugar decomposed and that the quantity of acid present at any one time during the time of the fermentation is not strictly proportional to the amount of sugar decomposed, for the reason that a part of the lactic acid resulting from the decomposition of the sugar is in all probability decomposed into other substances. According to this observer, 1,000 lactic bacilli decomposed in one hour an amount of sugar varying according to conditions from 0.00001 to 0.008 milligram.

The changes brought about in milk by micro-organisms are by no means confined to the production of lactic acid. In fact, as we have already seen, it is only by working with pure cultures of certain of the lactic acid organisms, such as the Streptococcus lacticus, etc., under proper conditions, that lactic acid alone is produced, and in the souring of milk, as this ordinarly takes place, a great many substances besides lactic acid are produced in larger or smaller amounts. Among these may be mentioned acetic, butyric, and succinic acids, alcohol and gaseous substances, such as hydrogen and carbon dioxide. In addition to these substances may be mentioned the production of small amounts of substances having characteristic odors usually of a disagreeable character.

It would seem from the recent work of Tissier and Gasching (20), carried on in Professor Metschnikoff's laboratory, that in the souring of milk, as this usually takes place, we have a more or less regular and definite sequence of changes, due to the growth and development in the milk of various species of micro-organisms which were always found by these observers to be present in the milk as it left the dairy. In the samples examined by them they found constantly bacteria and fungi. According to these authors the bacteria present in milk are divisible into two groups:

First. Mixed ferments, including the proteolytic mixed, such as Staphylococcus, rather rare, and the peptolytic mixed, such as Enterococci, B. coli, B. acidi paralactici, and B. lactopropylbutyricus.

Second. The simple ferments, including the simple proteolytic, such as Mesentericus, Subtilis, B. putrificus, and Proteus vulgaris; the simple peptolytic, such as Proteus Zukeri and B. foecalis alcaligenes.

The fungi are oidium lactis, rhizopus nigricans, and in one case a lactose yeast.

In sterilized milk these authors have found these organisms to produce the following changes:

The mixed ferments accomplish two principal fermentations in milk, the lactic and the butyric fermentations. The lactic fermentation is brought about by enterococcus, less actively by B. coli and most actively and vigorously by B. acidi paralactici, which possesses a high order of resistance. It produces chiefly dextrolactic acid. The butyric fermentation is accomplished by only one species, viz,

B. lactopropylbutyricus, which in order of sequence follows in the wake of the lactic fermentation. It is only dependent on it indirectly, however, since for the growth and development of this organism in milk neither lactic acid nor lactates are required, but a hexose which is formed from lactose by the bacteria immediately preceding the growth of the butyric ferment. Thus the butyric ferment depends only indirectly for its action on the lactic acid fermentation.

The simple ferments of milk have been found to peptonize and destroy the casein, but in symbiosis with the mixed ferments they are rapidly arrested in their action by the acid reaction of the medium and become powerless to effect those changes in milk which they ordinarily can accomplish. For the completion of these changes, therefore, the intervention of higher organisms is necessary. These are accomplished by the milk fungi, oidium lactis, and rhizopus nigricans.

The progress of the souring of milk has been found by these observers to be always the same. The mixed ferments develop rapidly, aided by the simultaneous action of the simple ferments. Enterococcus has been found to be the species predominantly producing inactive lactic acid, valerianic acid, and also always acetic acid. B. coli follows in its action, producing laevo lactic acid. Together these two organisms give an acidity to milk equivalent to 1.47 to 2 per cent of sulphuric acid. This degree of acidity arrests the action of the proteolytic ferments and brings on the coagulation or curdling of the milk. The B. acidi paralactici continues the destruction of the lactose, however, and gives rise to the true lactic acid fermentation, producing always dextrolactic acid. The medium having become favorable for its growth and development, the Bacillus lactopropylbutyricus sets up its characteristic fermentation, producing always inactive lactic and also propionic and butyric acids until a total acidity of 4 to 6 per cent in terms of sulphuric acid is reached. This degree of acidity arrests all bacterial action. The fungi, oidium lactis, and rhizopus nigricans then intervene, however, and by oxidizing the organic acids and lactose and by effecting a further destruction of the casein again favor the growth and multiplication of those organisms whose development has been momentarily checked. It also appears from the work of Tissier and Gasching that the simple ferments alone can bring about the decomposition of the casein and its ultimate derivatives. They have further observed that the bacteria ordinarily concerned in the souring of milk are not in any way directly responsible for the digestive disturbances which occasionally result from the use of milk as a food. Under certain conditions, however, they may act as predisposing causes, but the accidents of botulism are due, according to these authors, to special species of organisms differing from those which are ordinarily concerned in the souring of milk.

Quite recently Beyerinck (21) has again discussed the lactic fermentation of milk. He has found that temperature and oxygen pressure determine the nature of the autofermentation of milk. At temperatures below 40 C. the fermentation brought about by B. coli is replaced by a butyric acid fermentation, which, after lasting some time, is succeeded by a lactic acid fermentation. In good milk, even at 40° C., at which temperature gas-producing bacteria develop most rapidly, no gas is produced. This fact therefore forms the basis of a dairy test for judging of the purity of milk. He recognizes three forms of lactic acid fermentation depending on the temperature. At very low temperatures there occurs the slimy lactic acid fermentation, which, according to this author, is due to the smaller cell walls of the organism. At medium temperatures the common lactic acid fermentation predominates, this being caused by the lactococcus and at higher temperatures the lactic acid fermentation caused by the lactobacillus. Methods for isolating these organisms from milk are given, and also their morphological characteristics and their zymotic reactions. He, like other observers, has found the lactic acid ferment to be very variable.

Reference has already been made to the fact that the lactic acid fermentation of milk is used commercially in the manufacture of lactic acid. The lactic acid fermentation of milk is also turned to practical account in the manufacture of cheese. It has been shown by Epstein (22) that the ripening of cheese is due largely to the action of organisms which induce the lactic acid fermentation. Each particular kind of cheese is produced by the agency of special organisms which act chemically by means of enzymes and give rise to the peculiar odor and flavor of the cheese. These organisms are chosen both with regard to their power to induce the lactic acid fermentation and also with regard to the peculiar kind of cheese desired. Similar views regarding the ripening of cheese are held by Von Freudenreich (23). According to this author the lactic acid bacteria play the preponderating if not the exclusive rôle in the ripening of Emmenthaler cheese. Similarly Boekhaut and de Vries (24) have shown that cheese which does not contain the lactic acid bacteria does not ripen. On the other hand Chodat and Hofman-Bang (25) are of the opinion that the importance of the lactic acid bacteria in the ripening of cheese has been overestimated, and attribute the greater number of the changes occurring in this process to another organism—namely, tyrothrix.

In this connection it is interesting to note that Van Slyke (26) found that when only rennet is allowed to act on milk no cheese flavor is developed.

Abnormal fermentations of milk. Under ordinary circumstances milk usually undergoes the lactic acid fermentation. It turns sour and curdles and the production of lactic acid puts a stop, temporarily at least, to all other bacterial changes. Hence in normal milk it is only rarely that fermentation other than souring occurs. Under certain conditions, however, the milk becomes infected with a great variety of micro-organisms and various changes in its composition are brought about. By some authorities these have been called abnormal fermentations. As a result of these fermentations, alterations take place in the color, odor, and taste of the milk, and in some instances highly poisonous substances are produced. In this connection Burri and Dueggeli (27) have recently had occasion to examine four samples of milk in which such alterations had occurred. According to these authors sample (1) had the peculiarly disagreeable odor of Limburger cheese, sample (2) the odor of dogs, sample (3) a bitter taste, and sample (4) the odor and taste of Schabzieger cheese. These peculiar odors and tastes were found to be due to specific bacteria, which were isolated and their morphological characteristics determined by these authors.

Blue milk.- Under certain conditions a blue pigment may develop in milk as a result of peculiar changes set up by certain micro-organisms. While such milk is apparently harmless, it results from outside contamination and rarely if ever occurs in well-kept dairies. In one instance its production has been traced to some source of filth or uncleanliness and in some instances to a single cow. Its occurrence may be prevented by the adoption of cleanly methods and in case it has been traced to any particular cow by washing the cow's teats with a little weak acetic acid. It is of interest to note that blue milk is the first dairy infection definitely traced to bacteria. As early as 1841 Fuchs (28) traced the production of blue milk to the growth of a micro-organism. By using Koch's gelatin method Hueppe and Engling (29) succeeded in isolating the organism which produces blue milk. It was found by these authors to produce different colors when grown on different media, but in solutions containing ammonium lactate it was always found to produce a sky-blue color. Milk infected with this organism was always found to be alkaline, but the blue color only appears when the milk turns sour, as the result of lactic-acid fermentation, or when acid is added to the milk. J. Reiset (30) observed that in dairies of some localities a blue mold forms on the surface of cow's milk which has been allowed to stand. It has also been observed on the milk of ewes and goats. This mold was found to consist of mycelia containing immobile bacteria. The mold was found to grow only on milk having a distinctly acid reaction.

The chemical nature of the blue pigment was not determined. The organism ordinarily responsible for the production of blue milk is known as *Bacillus cyanogenes*. When grown in fresh milk the effect produced by this organism is very striking. During the first few hours however no change is noticeable. A certain amount of lactic acid seems to be necessary for the formation of the blue substance resulting from the growth of this organism. As the milk turns sour therefore blue patches appear, until finally these may be distributed throughout the whole of the milk, in such cases imparting to it a sky-blue color. Still other organisms besides *B. cyanogenes* seem to have the power of producing blue substances in milk.

Red milk.—According to Conn (31), red milk is by no means uncommon in the dairy. Ordinarily, however, the red color of such milk does not result from the growth of bacteria, but is due to the presence of blood in the milk resulting from injuries to the udder. Sometimes it results from the feeding of the cow on plants containing red pigment, such as the madder plant, etc.; more rarely from a peculiar fermentation induced by bacteria. Among the organisms known to produce this change in milk may be mentioned *Bacillus erythrogenes*, *B. prodigiosus*, and a sarcina. The production of red milk through the agency of bacteria is without practical significance.

Other color changes in milk.—Still other changes have been found to occur in milk and practically all of the pigment forming bacteria will develop their characteristic pigments in milk in the event that they gain access thereto. According to Conn (31) orange-colored milk, green milk, yellow milk, amber-colored milk, indigo milk, chocolate-colored milk, and black milk have all been described by bacteriologists. In all cases the pigment has been found to have been produced by bacteria. These have been isolated and their morphological characteristics determined. Ordinarily they are not normal infections, and hence are of no practical importance in dairying.

Slimy or ropy milk.—Under certain conditions, slimy, mucilaginous substances are produced in milk through the growth of certain organisms which impart to the milk a characteristic sliminess or ropiness. Milk possessing such properties is known as slimy or ropy milk. It often can be drawn out into long threads of exceeding fineness. For example, slimy milk has been obtained of such viscosity that it could be drawn out into threads 10 feet in length and of such fineness as to be scarcely visible. In certain countries slimy milk is esteemed a delicacy, and special methods have been devised for its preparation. Such is the case in Norway, where it is called by the natives "taetamoelk." In Holland also a special ferment is employed in the manufacture of Edam cheese, which has the power of rendering the milk slimy. The cheese made from such milk is said to ripen more rapidly and more evenly than cheese made without the use of this particular organism. This peculiar change in the consistency of milk has also been found to be due to bacteria and ordinarily, as it occasionally occurs in the dairy, is a source of great trouble and annoyance. Many bacteria seem to have the power of producing a slime in milk under suitable conditions. Ordinarily, however, this change is accomplished by one or two bacteria having a wide distribution in nature. Of these *B. lactis viscosus* (Adametz) seems to be the commonest organism of the kind found in Europe, and a similar organism, probably the same species, occurs in this country. It is a very hardy organism, and finds its way into the milk through the water supply of the dairy. From such a source the infection may become widely diffused and difficult to trace. However, it is an infection which, no matter how troublesome, can be eradicated through cleanliness, although in certain instances it may be necessary to resort to disinfectants. Among other organisms producing sliminess in milk may be mentioned *Micrococcus freudenreichii* and two forms of streptococci, one the source of the slimy ferment in Holland, the latter present on the leaves of Pinguicula, the latter being employed in Norway as the source of the ferment; and as pointed out by Beyerinck sliminess in milk may be produced by certain of the lactic-acid bacteria, especially by those growing at low temperatures. Slimy milk also results from a diseased condition of the mammary gland and is a common characteristic of garget. Nothing is known of the chemical nature of the substances causing the sliminess of milk.

Bitter milk. Freshly drawn milk has sometimes a bitter taste; in other instances it acquires such a taste on standing a few hours. The bitter taste of freshly drawn milk is sometimes due to the passage of bitter substances into the milk from the food of the cow, such as lupines. It may also be produced during the last stages of lactation. In those cases in which the bitter taste develops only after standing the cause thereof is to be sought in changes in the composition of the milk due to the action of certain organisms. A considerable number of organisms seem to possess the power of producing a bitter taste in milk; some of them after a short interval, others only after a longer one. Only the former are of any practical significance in the dairy, and among these may be mentioned a micrococcus, a cut of which is shown by Conn, and a bacillus described by Weigmann, both of which have the power of ruining the taste of freshly drawn milk in a few hours. The source of these organisms is difficult to trace. In one case cited by Conn the organism giving rise to this abnormal fermentation was traced to the milk ducts of a single cow.

According to Trillat and Sauton (32), bitter milk contains aldehydes and ammonia, and results from the simultaneous inoculation of fresh milk with a yeast producing aldehydes, and an ammonia-forming bacillus, B. Flügge, V.

The alkaline fermentation of milk.—It has been observed that boiled milk never turns sour by spontaneous fermentation. On the other hand, when boiled milk is allowed to stand at ordinary temperatures it gradually acquires an alkaline reaction, ofttimes a bitter taste, and finally curdles, yielding a soft, slimy curd. On further standing this curd gradually dissolves to form a somewhat clear liquid, and if the fermentation be allowed to proceed for a sufficient length of time a semitransparent liquid is obtained, having no resemblance to milk. As with the other fermentations of milk, a number of organisms are capable of causing the alkaline fermentation of milk, and a considerable number of substances are produced as the result of these changes. Among the substances found in milk which has undergone the alkaline fermentation may be mentioned the peptones, which are believed to be responsible for the bitter taste, leucin, tyrosin, and ammonia, which latter imparts to the liquid the characteristic alkaline reaction. Butyric acid is also formed in this fermentation. This, however, is at once neutralized by the ammonia present, and exists in the liquid in the form of ammonium butyrate.

Alcoholic fermentation of milk.—Among the abnormal fermentations of milk may be mentioned the alcoholic fermentation, which is accomplished by certain yeasts, aided in their action by certain species of bacteria. While the alcoholic fermentation of milk is abnormal in the sense that it never occurs in milk spontaneously, but must be induced by direct inoculation with certain ferments, it is employed in the production of certain milk beverages, such as koumiss and kefir, etc., which in certain countries are highly esteemed as articles of diet, and have in recent years come into more or less general use as food for invalids, etc. Koumiss, originally made by the alcoholic fermentation of mare's milk, is now made from cow's milk by the addition of cane sugar and yeast. The first action of the ferments is to hydrolyze the polysaccharides (cane sugar and lactose), producing the simpler sugars, glucose, levulose, and galactose, all of which are fermentable by yeast. Two changes then occur, the alcoholic fermentation, resulting in the production of alcohol and carbon dioxide, and the ordinary lactic acid fermentation, resulting in the production of lactic acid. Kefir, a similar beverage, originating in the Caucasus, is also made from milk by an alcoholic fermentation. The fermentation is carried out in leather bottles, and is started by means of "kefir grains," concerning whose origin but little is known. During the fermentation thus induced a considerable quantity of the ferment is produced, which is removed and dried in the sun, and thus new supplies of the kefir grains obtained. Struve (33) gives the following proximate chemical analysis of kefir grains dried at 100° C.:

	Per cent.
Water	11. 21
Fat	3. 99
Soluble peptone like substances	10. 98
Proteids soluble in ammonia	10. 32
Proteids soluble in caustic potash	30. 39
Insoluble residue	33. 11
	100. 00

The whole of the active matter of the ferment was contained in the insoluble residue. A microscopic examination of this showed it to consist of a mixture of yeast cells with *Bacterium dispora Caucasica* (Kern). In a few specimens leptothrix and oidium lactis were also present. According to this author, the yeast cells, which have been somewhat modified by their growth in leather bottles, are alone responsible for the peculiar kefir fermentation.

According to Vieth (34) milk sugar ordinarily does not readily undergo alcoholic fermentation with yeast. With kefir grains, however, a rapid alcoholic and lactic fermentation takes place. According to this author also the ferment of the grains consists of the *Bacillus dispora Caucasica* (Kern) and a modified form of the ordinary yeast, *Saccharomyces cerevisiæ*. According to von Freudenreich (35) the grains contain at least two species of bacteria and one species of yeast, which acting together produce the kefir fermentation. The bacteria effect the inversion of the milk sugar, after which a portion of the simpler sugar is converted into alcohol by the action of the yeast and another portion into lactic acid by the further action of the bacteria. The milk is curdled during this fermentation.

According to Martinand (36) milk undergoes alcoholic fermentation with a great many species of yeasts, especially if glucose and maltose be added, and coagulation of the milk occurs under these conditions even in the absence of acid.

Part II.—(4b) Milk Poisoning.—Galactotoxismus.

Of all foods milk is probably the most subject to contamination and change. Of the various forms of contamination to which it is liable the commonest is, as we have already seen, that which results from the introduction into the milk of lactic-acid-producing bacteria from various sources. These organisms accomplish those changes which are familiar to us in the ordinary souring of milk. While according to Stoakley (1) buttermilk is sometimes responsible for acute milk poisoning, it is the general opinion that sour milk but rarely gives rise to troubles of this character. Indeed, by a number of medical authorities sour milk is regarded as a very healthful beverage, by reason of the fact that the lactic-acid-producing bacteria tend by their growth in the intestine to lessen intestinal putrefaction, thereby diminishing the tendency to autointoxications from substances resulting from the growth of the bacterial flora normally present in the intestine. One the other hand, it not infrequently happens that fresh milk becomes contaminated with toxic substances, or with toxicogenic bacteria, in which event the milk may give rise to acute intoxications. The subject of milk poisoning has been chiefly studied by Vaughan and his associates, and to him we owe the term Galactotoxismus. In spite of all that has been done, however, the subject of milk poisoning is as yet but very imperfectly understood. Chiefly through the labors of Vaughan (2) and his coworkers, together with observations by Sonnenberger (3), Le Blanc (4), Baird (5), and others, it is now known that milk may acquire poisonous properties and become dangerous to health in essentially five distinct ways:

First. It may absorb metallic poisons from metallic vessels in which it has been allowed to stand. Attention has already been called to the fact that Golding and Feilmann (6) found copper in milk which had stood in contact with a broken copper coil. In this connection Baird (5) attributed an outbreak of milk poisoning to the preservation of milk in metal vessels, and pointed out that the substitution of earthenware vessels brought about a cessation of the trouble. Sonnenberger (3) has also observed that milk allowed to sour in vessels of copper, zinc, etc., is apt to contain soluble, poisonous salts of these metals.

Second. Through the elimination of poisonous drugs from the mother through the milk. As Sonnenberger (3) has pointed out, many drugs administered by the mouth appear in large quantity in the milk. Among such he cites ether, arsenic, alcohol, lead, colchicum, euphorbin, iodine, morphine, salicylic acid, hemlock, mercury, turpentine, antimony, veratrine, and a great variety of salts. He calls attention to the fact that all such milks are dangerous to children and young animals, and recommends that milk from cows receiving active drugs should not be allowed to be sold.

The excretion of drugs in the milk of nursing women has recently been made the subject of an exhaustive investigation by Bucura (7). According to this author, the number of drugs which have been found in human milk with certainty, following their administration to the mother, are very few. He himself investigated the excretion of forty of the drugs most commonly used on women during and after childbirth, and of these he found that only five or six could be recognized in the milk with certainty. These were aspirin, iodine, calomel (when taken internally), arsenious acid, potassium bromide, and probably also urotropin. From his own work and that of others, he gives the following list of drugs as having been found with certainty after their administration to the nursing mother: Iodine (following the external application of tincture of iodine or iodoform and the internal administration of iodides and iodothyrine), salicylic acid and salicylates, ether, mercury (following the use of mercury suppositories or after the interal administration of calomel), antipyrine, aspirin, arsenic, and bromides. A complete bibliography of the subject of the excretion of drugs in milk is given at the end of Bucura's article.

More recently Van Itallie (23) has found that after the injection of pilocarpine, physostigmine, morphine, arsenious acid, fluorescein, phenolphthalein and other drugs, tests for their presence in the milk were negative, while a trace of arsenic was found following the administration of Fowler's solution. Reijst-Scheffer (24) fed sodium iodide in solution to cows and determined the amount of iodine in the milk colorimetrically, with the following results: Iodine in the whole milk, 0.00178 to 0.00372 per cent; in the casein, 0.00012 to 0.00008 per cent; in the urine, 0.03 to 0.08 per cent. The milk fat contained no iodine.

Third. Through the elimination in the milk of poisonous substances contained in the food of cattle, especially the vegetable poisons of certain weeds which compose part of the diet of milch cows in many localities. According to Sonnenberger (ibid.) deleterious cattle feed is very common. Clover fields around Worms (Germany), for example, have been found to contain 30 to 40 species of more or less poisonous plants, 15 of which are very poisonous. According to this author, these poisons pass into the milk if such plants are eaten by the cows; and these poisonous substances are not destroyed by boiling the milk. He found in harmony with these ideas that the season for infantile diarrheas around Worms corresponds not with the hot season, but with the season most favorable to the growth of weeds, viz, a cold, wet summer. According to Sonnenberger, the feeding of milch cows with vegetable refuse, such as potato tops, rotten apples, moldy hay, etc., tends also to poison the milk.

Fourth. It has been demonstrated that milk may acquire toxic properties as a result of a diseased condition of the mother. According to Michelazzi (See Le Blanc (4)), the milk of a tuberculous animal contains a tuberculous poison, which is not entirely destroyed by heating to 100 C., and that the milk of such animal, when sterilized at 100 C., causes a slow, chronic intoxication, and that the milk of a tuberculous mother is toxic to the children. Le Blanc has pointed out that the milk of cows in heat (*les vaches taurelières*) has a strong, cheesy smell, and a salty, bitter taste. It alters rapidly even when kept in sterile tubes, and causes gastro-intestinal disturbances in young animals. The toxic effect of the milk and milk products of " nymphomanous " cows is even more marked.

Lawrence (reference 79, Part I) has also recently observed the passage of typhoid bacilli into the milk of a nursing woman ill with typhoid fever.

Fifth. As shown by Vaughan and others (2), highly toxic substances are produced in milk by bacteria. The earlier investigations on the subject of bacterial poisons in milk and milk products were confined almost entirely to poisonous cheese, the poisonous properties of which were formerly ascribed to various fatty acids. In 1852, however, Schlossberger (see Vaughan & Novy, ibid.), from experiments with pure fatty acids, demonstrated that these substances are not sufficiently toxic to account for the highly toxic nature of poisonous cheese. In 1883 and 1884 an epidemic of cheese poisoning occurred in Michigan, which led Vaughan and his students to an exhaustive investigation of the subject. The outcome of these studies was the isolation from poisonous cheese, in 1884, of a crystalline substance, to which Vaughan gave the name of tyrotoxicon, and which was believed by him to be a diazo derivative of benzene. Chemically it was found to be very unstable, its aqueous solution decomposing when heated to 90° C. Tyrotoxicon has since been isolated, in many instances, from poisonous cheese by other investigators. It has also been detected in poisonous milk. In 1886 Newton and Wallace (8) found the poison in a milk supply at Long Branch which had seriously affected a number of persons. In 1887 Firth (9), an English army surgeon, isolated it from the milk which had poisoned the soldiers of a garrison in India where he was stationed, and in the same year Vaughan (10) investigated a number of cases of violent milk poisoning occurring at Milan, three of which had resulted fatally. Fresh milk, inoculated with the vomit, stomach contents, or an aqueous extract of the intestines, gave, after standing twenty-four hours at 25°-30° C., a sufficient amount of tyrotoxicon to enable these investigators to recognize nitrogen and phenol among the products of its decomposition, the latter being recognized by precipitation with bromine water and by other well-known tests. In these cases the coroner's jury, before whom this evidence was submitted, rendered a verdict of death from poisoning by tyrotoxicon. Cannnan (11) reported 23 cases of milk poisoning attributed to tyrotoxicon, and Kinnicutt (12) isolated the poison from milk which had stood in unclean vessels for some time. Vaughan and Novy (13) and others found tyrotoxicon in poisonous ice cream, and still others have obtained it from custards and other desserts prepared from milk or cream. Indeed it would appear from these investigations that any foodstuff prepared from milk is liable to contain this poison. In his later writings on the subject, however, Vaughan (14) takes the view that tyrotoxicon is not the only poison in poisonous cheese and milk products. According to this author it is probably not present in all specimens of poisonous cheese, and it is probably not the most important poison of poisonous cheese. Others are also of this opinion. For example, Dokkum (15) by the methods used by Vaughan in the isolation of tyrotoxicon obtained from poisonous cheese a substance similar to curare in its action, five milligrammes of which killed frogs in thirty minutes. For this poison this author proposed the name tyrotoxin to distinguish it from tyrotoxicon. Nothing is known regarding its chemical composition. Lepierre (16) isolated a base having the composition $C_{16}H_{23}N_2O_4$ from poisonous cheese, which caused diarrhea in animals when administered by the mouth. During the course of their own investigations, Vaughan and Novy (17) were unable to detect tyrotoxicon in certain samples of cheese known to have produced poisoning. From some of these samples they obtained a poisonous albumin. It gave the biuret test. It was found not to be a globulin nor a peptone. On the other hand, certain bacteria obtained from poisonous milk and cheese developed on culture media poisons which, according to Vaughan, are probably related to neurin.

It has also been shown that milk and milk products may also contain a large number of bacteria each of which produces its peculiar toxin (18). This, according to Novy (19), is especially the case with the Enteritidis group of bacteria, which by their growth do not curdle the milk, but render it somewhat transparent. According to Vaughan, the summer diarrheas of children are not due in all cases to a specific micro organism, but to the poisons elaborated in milk by many different bacteria. Such diseases are found almost exclusively among children that are artificially fed, and they occur chiefly in the hot weather, for the reason that a high temperature is essential to the growth and wide distribution of these toxicogenic organisms. To Fluegge (20) we are indebted for some of the most valuable contributions to our knowledge of the toxicogenic bacteria of milk, especially the peptonizing bacteria. By this author 12 such species were isolated and studied. Of these, 3 species were found to develop poisonous substances. Cultures of No. 1 in subcutaneous doses of 0.5 cubic centimeter were found to kill mice. When milk containing this organism was fed to dogs similar disturbances set in in about one hour. Milk cultures of bacillus No. 3 produced diarrhea in puppies, followed in one case by death on the third day. The filtered culture of bacillus No. 7, after concentration to one-fifth of its original volume, caused death in mice and guinea pigs in six to twelve hours, and even the unconcentrated milk culture of this organism acted powerfully when fed to puppies. In market milk Fluegge frequently found these poisonous peptonizing bacteria in practically pure culture. The investigation of the peptonizing bacteria of milk has been continued by Luebbert and also by Vaughan. According to Luebbert (21), these organisms are widely distributed. They have been found to act only on the proteids of the milk, the fat and milk sugar contained in the milk remaining undiminished. He also found the milk cultures of these organisms to be highly toxic. When fed on such milk, guinea pigs died after four days, and puppies after the fourth, fifth, and sixth day, following severe diarrheas. On the other hand, a full-grown dog ate of the milk freely without any bad effect, thus showing that age affords some protection against milk poisoning. Luebbert's results on the toxicogenic peptonizing bacteria of milk have been confirmed by Vaughan (22). According to this author the organisms responsible for cholera infantum are truly pathogenic in that they produce a definite chemical poison, the absorption of which is followed by the symptoms of the disease, and in order to explain the great susceptibility of infants to milk poisoning and the comparative immunity of the adult he has advanced the view that the great susceptibility of children to such intoxications is due to the ease and readiness with which casein is absorbed by the mucous membrane of the intestine of children, and that the casein carries along with it the bacterial cells containing this poison. In the adult, on the other hand, the digestive powers of the stomach are increased and intestinal absorption modified to a corresponding degree. At present practically nothing is known regarding the precise chemical nature of these bacterial poisons, and, as already pointed out by Novy (19), investigations pertaining to a more exact study of the toxicogenic micro-organisms of milk and their poisonous products belong to the future of medical and chemical research. I have been informed by Doctor Vaughan that nothing of any practical importance has been added to our knowledge of the milk poisons during the last few years.

PART III.—CHEMICAL STANDARDS FOR THE CONTROL OF THE SALE OF MILK.

For a number of years the sale of milk in various cities throughout the world has been regulated by law and various chemical standards regulating the sale of milk have been proposed, based on the results of large numbers of analyses of milk in various countries. I am indebted to L. A. Rogers, Acting Chief of the Dairy Division, Bureau of Animal Industry, U. S. Department of Agriculture, for the following compilation of United States and State standards for milk and dairy products. It will be observed that this compilation was published in 1905.

[U. S. Department of Agriculture, Bureau of Animal Industry—Circular No. 74. D. E. Salmon, D. V. M., Chief of Bureau.]

WASHINGTON, D. C., *April 1, 1905.*

In the table following, prepared under the supervision of Ed. H. Webster, Chief of the Dairy Division, are given the standards for dairy products as proclaimed by the Secretary of Agriculture and as established by law in the several States, so far as obtainable, and revised to date.

The percentages stated represent miniumum standards in all cases unless otherwise expressed. States not named are understood to have no laws prescribing standards for dairy products.

D. E. SALMON,
Chief of Bureau of Animal Industry.

Approved:
JAMES WILSON,
Secretary of Agriculture.

United States and State standards for dairy products, 1905.

States.	Milk.			Skim milk.	Cream.	Butter.	Cheese.
	Total solids.	Solids, not fat.	Fat.	Total solids.	Fat.	Fat.	Fat.
	Per cent.	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per cent.*	
United States[a, b]	12	8.5	3.25	9.25	18	82.5	Full cream, 50 p. c. of the total solids to be fat.
California					(c)		Full cream, 30 p. c. fat; half skim, 15 p. c.; skim from skim milk. Fancy excepted.
Colorado							Full cream, 35 p. c. total solids to be fat; skim, fat less than 35 p. c. of total solids.
Dist. of Columbia		9	3.5	9.3	20	83 Not over 12 p. c. water or 5 p. c. salt	
Georgia		8.5	3.5				
Hawaii	11.5		2.5				
Idaho		8	3		18	82.5	Full cream, 30 p. c. fat (fancy excepted; skim, less than 30 p. c. fat; less than 15 p. c., sale prohibited.
Illinois[d]			3		(e)	80	Whole milk, 48 p. c. total solids to be fat.
Indiana		9	3			80 Maximum water, 15 p. c.; salt, 6 p. c.	Skim, minimum fat 10 p. c.

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[a] See proclamation of the Secretary of Agriculture, "Standards of Purity of Food Products," Office of the Secretary, Circular No. 10, November 20, 1903.

[b] Condensed milk, 28 per cent milk solids, of which one fourth must be fat.

[c] Cream containing thickener must be labeled.

[d] Condensed milk must contain not less than 8.5 per cent fat; evaporated cream containing less than 15 per cent fat must be labeled "An unsweetened condensed milk."

[e] Coffee cream shall contain at least 15 per cent fat, and whipping cream at least 22 per cent fat.

United States and State standards for dairy products, 1905—Continued.

States.	Milk.			Skim milk.	Cream.	Butter.	Cheese.
	Total solids.	Solids, not fat.	Fat.	Total solids.	Fat.	Fat.	Fat.
	Per cent.	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per cent.*	
Iowa	12.5		3		15		
Kentucky	12		3		15	80	Skim, less than 10 p. c. fat.
Maine	12		3				
Maryland[a]	12.5		3.5				
Massachusetts	13	9.3	3.7	9.3			
April-September	12	9	3				
Michigan	12.5		3				
	Sp. grav. 1.029–33			Sp. grav 1.032–37			
Minnesota	13		3.5		[b]20	Maximum water, 16 p. c.	Full cream, 45 p. c. total solids to be fat, skim, fat less than 45 p. c. of total solids.
Missouri							Full cream, from 3 p. c. milk fat; skim, from milk less than 3 p.c. fat.
Montana	11.5		3		15		
Nebraska			3		15		
New Hampshire	13	9.5	3.5	9			
April-September	12		3				
New Jersey	12						
New York[c]	12		3				Skim, from skim milk.
North Carolina[d]	12	8.5	3.25		18	82.5	Full cream, 50 p. c. total solids to be fat; skim, from skim milk; cream cheese, milk 6 p. c. minimum fat.
North Dakota	12		3		15		Skim, from skim milk.
Ohio[d]	12		3			80	Full cream, 30 p. c. fat; skim, less than 30 p. c. fat.
May-June	11.5						
Oregon	12	9	3	9 Sp. grav. 1.038	20	Not over 14 p. c. water.	Full cream, 30 p. c. fat; half skim, 15 to 30 p. c.; quarter skim, 7½ to 15 p. c.; skim, less than 7½ p. c. Fancy excepted.
Pennsylvania	12		3	8			Full cream, 32 p. c. fat; three-fourths cream, 24 p. c. fat; one-half cream, 16 p. c. fat; one-fourth cream, 8 p. c. fat; skim, below 8 p. c. fat. Fancy, less than [illegible] pounds, excepted.

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[a] Condensed milk must contain the equivalent of 12.5 per cent of milk solids in crude milk of which 3.5 per cent shall be fats.

[b] No thickener allowed.

[c] In New York, Ohio, and Wyoming the milk solids of condensed milk must be in quantity the equivalent of 12 per cent of milk solids in crude milk, of which solids 25 per cent shall be fat.

[d] Condensed milk must contain 28 per cent milk solids and 7 per cent fat.

United States and State standards for dairy products, 1905—Continued

States.	Milk.			Skim milk.	Cream.	Butter.	Cheese.
	Total solids.	Solids not fat	Fat.	Total solids.	Fat.	Fat.	Fat
	Per cent	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per cent*	
Porto Rico	12	.....	3			80 Maximum water, 16 p.c.; salt, 6 p. c.	Full cream, 30 p. c. fat, one-half skim, 15 p. c. fat, skim, 10 p. c.
Rhode Island	12	..	2.5	...		..	
South Carolina	..	8.5	3				
South Dakota	13	..		...	18	82.5	Full cream, 50 p. c. of the total solids to be fat; skim, fat less than 50 p. c.
Utah ..	12	..	3	9 p. c. solids, not fat.	20	80	Skim, 7 to 11 inches in diameter; minimum height, 9 inches.
Vermont .	12.5	9.25	..	....	....	...	
May and June .	12	....	.	....	....	....	
Washington	..	8	3	... ...	18	....	Full cream, 30 p. c. fat; skim, 15 p. c. fat. Fancy excepted.
Wisconsin	..	.. ...	.	..	...	....	Skim, 10 inches in diameter, 9 inches height.
Wyoming ..	12	....	2.4	.. .. .	....	80	Skim, less than 20 p. c. fat.
May and June	11.5	....		.. ..	....	....	

Condensed milk must contain 28 per cent milk solids and 7 per cent fat.

At the Eleventh Annual Convention of the Association of State and National Food and Dairy Departments, held at Jamestown, July 15-19, 1907, Mr. P. M. Harwood (1), general agent Massachusetts dairy bureau, read a paper entitled "Has the milk standard outlived its usefulness?" He called attention to the fact that on account of the rigid requirements regarding the composition of milk offered for sale in the State of Massachusetts a good many milk producers are being gradually driven from the business for the reason that while milk prices are gradually becoming higher in the cities, the farmer or milk producer does not receive a proportionate amount of the profit accruing from the increased price nor an amount sufficient to compensate him for the trouble and expense growing out of the enforcement of laws regulating the milk standard. He points out that at a recent discussion of the question of the milk standard before the committee on agriculture of the Massachusetts legislature many interesting facts were brought out. It developed at these hearings that the standards now in force, viz, 13 per cent total solids, 3.7 per cent fat, and 9.3 per cent solids not fat, in winter, and 12 per cent total solids, 3 per cent fat, and 9 per cent solids not fat, in summer—are working a hardship on the farmers, and that indirectly they are not protecting the consumer; that milk contractors and peddlers were using it to their pecuniary advantage, and that the prosecuting officers throughout the State were not rigidly enforcing the law. The author reached the conclusion, therefore, that either the milk standard should be abolished altogether and milk sold upon its merits, or, that if a standard is to be maintained, it should be uniform throughout the United States. On account of the very large amount of data on the chemical composition of milk at present available in State and municipal departments and agricultural experiment stations, etc., such a standard could probably be equitably adjusted. Indeed, the attempt has been made to do so in establishing the United States milk standard governing the sale of milk under the laws governing interstate commerce. This standard requires a milk to contain 3.25 per cent of fat and 8.5 per cent solids not fat, and, as may be seen from the tables of State and national milk standards given on page 378, it is lower than many of the State standards. According to the secretary of the association of State and national food and dairy departments, the United States standard is being made the basis of standards for all the States.

In this connection it is of interest to note that certain high-class dairies throughout the country are prepared to furnish milk of any composition desired, and infants' milk according to the physician's prescription.

PART IV.—ADULTERATIONS OF MILK.

Like many other foodstuffs, milk is subject to many adulterations. These consist (1) in the removal of the cream (skimming) or the addition of skim milk, (2) addition of water (watering), (3) addition of thickening agents, (4) the addition of coloring matters, (5) the addition of certain substances with the view of altering the taste of milk and increasing the total solids, (6) the addition of preservatives (antiseptics). The commonest forms of adulteration are skimming, watering, and the addition of artificial coloring matters and preservatives, the addition of thickening agents, such as chalk, calves' brains, starch, glycerin, etc., having almost passed out of vogue among farmers and dairymen. Indeed it is doubtful whether this (3) form of adulteration was ever practiced to any considerable extent (see Leach, 1).

Skimming.—This form of milk adulteration is probably practiced among farmers and dairymen to a considerable extent. As its name indicates, it consists in the removal, by means of a separator or otherwise, of a part of the cream. Obviously, skimmed milk contains a smaller percentage of milk fat than normal milk, and it was with a view of correcting and controlling this particular form of adulteration and watering that laws have been enacted in many countries and in many States throughout our own country fixing the amount of milk fat which a milk offered for sale should contain. It will be seen from the compilation of milk standards given on page 378 that the amount of fat required in different States varies from 2.4 to 3.5 per cent. All things considered, it seems reasonable to require that all milk offered for sale should contain at least 3.25 per cent of fat, although it should be borne in mind, of course, that unadulterated milk, especially of certain breeds of cattle, sometimes contains less than this amount of milk fat. The color of skimmed milk is also more or less characteristic, tending more to dead white or bluish white than normal milk, which is distinctly yellowish white in color.

Watering. —The addition of water to milk is probably the commonest practice in milk adulteration. Obviously, this is done in order to increase the output of the dairy. The effect of watering is to alter the physical properties and chemical composition to a greater or less degree, depending on the quantity of water added. The addition of water to milk has been found to lower the specific gravity and raise the freezing point of milk. It also lowers the index of refraction and probably the viscosity. It causes a diminution in the amount of fat, total solids, and ash. Ordinarily it is not a difficult matter to determine whether a given sample of milk has been watered. This is done by comparing its specific gravity and refractometer reading, together with the amounts of fat, total solids, and ash, with those of normal milk or with standards which have been based upon the results of thousands of analyses and years of experience with the milk of different herds of dairy cows and that produced in different countries. In the detection of watered milk advantage is also taken of the fact that natural waters frequently contain substances not ordinarily present in milk, such as nitrates and nitrites. If these substances are found in a sample of milk the chances are that water has been added to it. It has been proposed by Steinegger (2) to employ the aldehyde value as a means of detecting the addition of water to milk. The aldehyde value for normal milk in Soxhlet-Henkel degrees varies between 5.8° and 8.5° and is lowered by the addition of water to milk, but not by the removal of fats. According to Commanducci (3) the watering and skimming of milk may be determined by the lowering of what he proposes to call the index of oxidation of milk. This he determines by means of tenth-normal potassium permanganate in acid solution. The number of cubic centimeters of potassium permanganate solution required to oxidize 1 cubic centimeter of milk is what this author calls the index of oxidation. This has been found to be different for the milk of different animals, but practically constant for the normal milk of any particular animal species. He gives the following values for the index of oxidation of the milk of the following animals:

Cow	50-52	Ass	55-58
Goat	44-46	Woman	53-60
Sheep	43-48		

He also finds that the value of the index of oxidation of cow's milk diminishes with the amount of water added, and also with skimming. Thus the index of oxidation of cow's milk containing 50 per cent of water was found to be 25, and that of skimmed milk 40 to 42.

According to Atkins (4), determinations of the freezing point and of the specific gravity of milk are sufficient to show whether water has been added or fat removed.

The addition of water to milk is not only a fraudulent practice and one which as such should be condemned, but it may frequently be a serious menace to the public health. Atlee (5) has pointed out that impure water is one of the most frequent sources of milk pollution. This pollution may occur either through the use of impure water for purposes of adulteration or as the result of washing the milk containers and utensils in polluted water. As is well known, milk is one of the best possible culture media for the growth of microorganisms, especially for many of the pathogenic bacteria. It is conceivable, therefore, indeed it is a well-known fact, that the introduction of a few pathogenic organisms into milk through the addition of impure water will under certain conditions give rise to a fluid containing countless numbers of such organisms. In this way the adulteration of milk with water may give rise to a widespread dissemination of various infections, especially typhoid fever, diphtheria, scarlet fever, etc. Aside therefore from the fraudulent aspect of the practice, the adulteration of milk with water, from any and every source, as frequently happens, becomes a matter of serious concern, and of all fraudulent and uncleanly practices resorted to in the handling and sale of milk this and the uncleanly methods of handling milk are the two which should be most vigorously combated and condemned.

Thickening agents.—As indicated above, the adulteration of milk through the use of thickening agents, such as chalk, calves' brains, glycerin, etc., has largely passed out of vogue. Indeed it is doubtful whether any of these substances were ever used to any considerable extent, despite traditions to the contrary. According to Van Slyke (6) gelatin and sucrate of lime are used to some extent to give a greater consistency to cream. In this connection Babcock and Russell have recommended the use of sucrate of lime for restoring the consistency of pasteurized cream. (See Leach (1), p. 156.)

Condensed unsweetened skim milk has also been employed as an adulterant, with the object of increasing the consistency and raising the total solids of a skimmed or watered milk.

The addition of substances with the view of altering or disguising the taste of milk or of increasing the total solids.—Milk is sometimes adulterated by the addition of certain substances intended to alter or disguise the taste of milk. These are sodium carbonate and bicarbonate, cane sugar, and saccharine. Sodium carbonate and bicarbonate are sometimes added to sour milk with the view of neutralizing the lactic acid and preventing or delaying the separation of the curd. Cane sugar is added in order to increase the amount of total solids in milk impoverished by watering, and also to increase the sweet taste and thereby disguise any slightly sour taste which old milk may possess. Saccharine is sometimes added to milk for the same purpose. It not only increases the sweet taste of milk, but probably also acts as a mild antiseptic. While all of these substances are probably harmless in the amounts in which they are employed in milk (certainly the addition of cane sugar can ordinarily do no particular harm), the practice of adding these substances to milk is to be condemned, mainly on the ground that they are rarely used except to conceal deficiencies in the quality of the milk itself, thereby enabling the dairyman to palm off on the consumer milk which ordinarily would not be found acceptable.

Coloring matters.—Milk is sometimes adulterated by the use of artificial coloring matters. The principal object to be accomplished by the use of these colored substances is to conceal other forms of adulteration, such as skimming and watering, and to make the milk appear richer than it really is. It has been pointed out in the foregoing that skimming and watering cause an alteration of the color of milk as compared with normal milk. Generally milk that has been skimmed or watered is more whitish in color than milk containing the normal quantity of cream. In order to conceal these deficiencies in the color of milk so adulterated various artificial coloring matters are added in order to bring the milk up to the color of normal milk. Among the coloring matters which have been employed for this purpose are annatto, certain of the yellow and orange-colored azo dyes, caramel, etc. Generally speaking, the adulteration of milk with these artificial coloring matters is in itself of minor importance, inasmuch as they are used in very small quantities and the coloring matters ordinarily employed in the artificial coloring of milk have been found to be harmless. The fact, however, that they are employed mainly with a view of concealing other more dangerous adulteration, such as the addition of water to the milk, puts the addition of artificial coloring matters to milk in the class of dangerous adulterations. In this connection it has been pointed out by Winton (7) that in the examination of a foodstuff for artificial colors the chemist ofttimes encounters the difficulty of distinguishing a harmless from a poisonous color. As a rule it is an easy matter to determine when a synthetic color is present, but very difficult ofttimes to determine its precise nature. Then again, as pointed out by Tolman (8), the coal-tar colors are frequently contaminated with powerful mineral poisons, such as arsenic, copper, tin, lead, and zinc, which are introduced as impurities in the process of manufacture. It has been established further that many of the coal-tar dyestuffs are poisonous and that still others not very actively poisonous are nevertheless sufficiently so to interfere with the action of the digestive ferments. For example, Houghton (9) found that annatto diminished the digestibility of casein and egg albumen by pepsin. For further information regarding the toxicity of the coal-tar dyes the following authorities should be consulted: Weyl (10), Weber (11), Winogradow (12), Gudeman (13), Chlopin (14), and Meyer (15).

Preservatives.—We have seen that milk is subject to many changes, principally those resulting from the life and growth therein of micro-organisms. Indeed, it is one of the most perishable of foodstuffs, and it is only by exercising the most scrupulous cleanliness in the handling of it and by keeping it at low temperatures, generally below 50° F., that it can be preserved a sufficiently long time to be delivered to the consumer in a fresh condition. This has resulted in the practice on the part of dairymen of adding to the milk small amounts of various antiseptics and germicides, which are supplied to the trade under the general name of milk preservatives. The effect of such substances is to destroy or at least hinder the growth of all micro-organisms which the milk may contain, and thereby retard the souring of the milk; and to prevent or at least delay the lactic-acid fermentation of milk is the principal object to be attained through the use of such substances. Among the various substances which have been employed as milk preservatives may be mentioned the following:

Common salt, sodium bicarbonate, formaldehyde (solutions of which are supplied to the dairyman under the trade name of "Freezine"), borax and boric acid (solutions of the latter once sold under the name of "Aseptine"), salicylic acid, benzoic acid, hydrogen peroxide, certain fluorides, potassium dichromate, etc. Of these substances formaldehyde, boric acid and borax, and sodium bicarbonate have probably been the most frequently employed as milk preservatives. In certain localities in Europe the addition of alkali chromates to milk was at one time a common practice, and Budde (16) has proposed a method for the sterilization of milk by the action of hydrogen peroxide at a moderately high temperature, viz, 52° C.

It is doubtful, however, whether the method ever found any very extensive application. According to Leach (17) salicylic and benzoic acids are now rarely used as milk preservatives. Salicylic acid in quantities sufficient to preserve affects the taste of the milk. Richmond (18) found a new food preservative to consist of acid potassium fluoride, KHF_2.

As a general thing these substances are employed only in small amounts, and at present there is considerable difference of opinion as to what effect these various substances in the small amounts usually present in milk and other foodstuffs exert upon the human system. Thus, according to Trillat (19), formaldehyde renders the casein of milk more or less indigestible, and a further objection to its use is that part of it remains unaltered in the various foodstuffs with which it is admixed, and being absorbed as such by the system may act injuriously on the digestion. On the other hand, Rideal and Foulerton (20) have observed that formaldehyde at a dilution of 1 : 50,000 or 0.05 per cent of boric acid and borax will preserve milk twenty-four hours, and that these amounts of these substances have no effect on the peptic and pancreatic enzymes, while this quantity of boric acid greatly retards the diastatic power of saliva, the formaldehyde having much less effect. Experiments with kittens, rabbits, and guinea pigs proved, according to these observers, that the amount of formaldehyde required to preserve milk has no effect on their proteid metabolism. Fish were not affected in six days in water containing 1 part of formaldehyde in 50,000 parts of water, and frogs stood a concentration of 1 : 20,000 without injury for two hours. The conclusion drawn by these writers from their investigation is that the quantities of these substances necessary to preserve milk twenty-four hours have no appreciable effect on the digestibility of the milk, and that in these quantities formic aldehyde and boric acid interfere less with the pancreatic digestion of casein than tea, claret, and Worcester sauce. Formaldehyde, 1 : 50,000, does not appear to have any injurious action upon animal tissues, or on nutrition. On the other hand, Otto Hehner (21) has criticized the experiments by Rideal and Foulerton on the ground that they were not properly controlled, and this author seems inclined to believe from the results obtained that these substances, in the quantities employed, were in reality injurious to the animal organism. T. M. Price (22), working in the Biochemic Division of the Bureau of Animal Industry, U. S. Department of Agriculture, has made a valuable contribution to this subject. He has studied the effect of some food preservatives on the action of the digestive enzymes, especially the effect of formaldehyde on the preservation of milk and the effect of this substance on the digestibility of the milk by the digestive enzymes in vitro and in the stomach of the calf. The following are the more important conclusions which he has drawn from these investigations:

(1) Formaldehyde in the proportion of 1:20,000 preserves the milk for forty-eight hours.

(2) Formaldehyde in milk in the proportion of 1:10,000 does not interfere with the digestion of the milk when it is fed to calves.

(3) Upon feeding calves through a long period with milk preserved with formaldehyde the calves remained healthy and gained in weight.

(4) Formaldehyde added to milk in the proportion of 1:2,500 or less has no effect on the activity of the fresh enzymes, rennet, pepsin, pancreatin, and steapsin, in vitro.

(5) Formaldehyde added to starch in the proportion of 1:2,500 or less has no effect on the conversion of the starch into sugar by the enzymes ptyalin and amylopsin, in vitro.

(6) Formaldehyde added to milk in sufficient quantity to preserve the milk for forty-eight hours—i. e., 1:20,000—does not materially interfere with the action of the enzyme galactase, in vitro.

(7) Formaldehyde added to milk in the proportion of 1:20,000 prevents the development of the more common bacteria found in milk and when added in the proportion of 1:1,560 it kills these bacteria.

(8) Formaldehyde may be added to milk in sufficient quantities to preserve the milk and to prevent the development of some of the more common bacteria—i. e., 1:10,000—and still have no deleterious effect on the digestibility of the milk for calves.

(9) Formaldehyde should never be fed to calves as a milk preservative stronger than 1 part of formaldehyde to 10,000 parts of milk.

According to Price the results obtained by the majority of investigators who have experimented with formaldehyde are of no value, inasmuch as at least the majority of them employed formaldehyde solutions varying in concentration from 1:25 to 1:2,000, these quantities being very much larger than the quantities of formaldehyde used in the preservation of milk in practice. At the close of his article Price gives the following bibliography of the subject:

(1) Salkowski u. Hahn, Pflüger's Archiv., Bd. LIX u. LXIII; Moraczewski, Zeitschr. f. physiol. Chem., B. XX.

(2) Babcock and Russell, Wis. Ann. Rept. Ex. Stat., Vol. XIV, 1897, p. 161.

(3) Snyder, Minn. Ex. Stat. Bull. No. 74.

(4) Babcock and Russell, Wis. Ann. Rept. Ex. Stat., Vol. XV, 1898, p. 77.

(5) Van Slyke, Rept. N. Y. Ex. Stat., Vol. XX, 1901, p. 165.

(6) Loew, Ann. Agronom., Vol. XCVIII, p. 416; Pottevin, Ann. de l'Inst. Pasteur, 1897, p. 807; Symons, Jour. Am. Chem. Soc., Vol. XIX, 1897, p. 724; Foulerton, Lancet, Vol. XI, 1899, p. 1578; Bliss and Novy, Jour. Ex. Med., Vol.

IV, 1899, p. 60; Halliburton, Brit. Med. Jour., Vol. XI, 1900, p. 1; Rideal and Foulerton, Pub. Health, 1899, p. 554.

(7) Cripp, Analyst, Vol. XXII, 1897, p. 182; Allen, Lancet, Vol. I, 1896, p. 1516; Ringer, Jour. of Phys., 1895, p. 425; Chittenden, Diet. and Hyg. Gaz., 1893, p. 25; Mayberry and Goldsmith, Chem. Centralbl., 1898, p. 69; Leffmann, Jour. Franklin Inst., 1899, p. 103; Weber, Jour. Am. Chem. Soc., 1902, p. 4; Chittenden and Gies, Am. Jour. Phys., 1898, p. 1; Tunnicliffe and Rosenheim, Jour. Hyg., 1901, p. 168; Forstus, Archiv. of Hyg., Vol. XI, 1884, p. 75; Liebreich, Vierteljahrsschr. gericht. Medizin, 1900, p. 83.

Reference has already been made to the fact that Trillat (19) found that formaldehyde renders the casein of milk more or less indigestible. In this same connection Pottevin (23) has observed that formaldehyde retards the coagulation of milk by rennin. Further experiments along this line have been made by Bliss and Novy (24). These authors have confirmed the conclusions of Pottevin regarding the influence of formaldehyde on the coagulation of milk by rennin and have found that under the influence of formaldehyde the caseinogen of milk is rapidly altered in such a way that either the rennin coagulation takes place only very slowly or not at all. Thus if formaldehyde in the proportion of 1:500 be allowed to act on milk for a few hours the milk is not coagulated on the addition of rennin. On the other hand, they observed that the rennin itself is not readily destroyed by formaldehyde, so that the delay or hindrance of the rennin coagulation of milk by formaldehyde is evidently due in some way to an alteration in the composition or properties of the caseinogen. Similar experiments on the action of formaldehyde on the digestive ferments have been made by Halliburton (25). He observed that 0.5 per cent of formaldehyde renders gastric digestion almost impossible, and 0.05 per cent delays it considerably. With 0.1 per cent formaldehyde no pancreatic digestion of fibrin occurs in twenty-four hours, and dilute solutions of the aldehyde delay the pancreatic digestion of starch. He also confirms the deleterious effects exerted by formaldehyde on the rennin coagulation of milk.

Wiley and his coworkers have also studied the effect of formaldehyde on the health of man. The results of this investigation, however, have not yet been published.

Concerning the toxic effects of boric acid and borax there is also the greatest difference of opinion among those who have made a study of the subject, and more recent investigations, despite their exhaustive character, have tended by no means to reconcile these opposing views, but if anything to accentuate them. For example, J. Neumann (26) found that only very large doses of boric acid can cause death by gastroenteritis or from its effects on the nervous or muscular systems. He therefore recommended it for the preservation of milk. According to Cyon (27) borax diminishes proteid metabolism, but all that can be learned from his work on the subject is, that metabolism and assimilation were not seriously interfered with by borax in the quantities administered. Gruber (28), on the other hand, found that borax increases proteid metabolism and concludes that borax exerts no unfavorable influence on the assimilation of food. According to this author, no harmful effect followed a maximum dose of 20 grams. Forster (29), from his studies on the applicability (verwendbarkeit) of boric acid as a food preservative, concludes that, while boric acid is without influence on proteid metabolism, the continuous administration of small amounts of it in food is not without its drawbacks so far as the health of the individual is concerned, and that its use as a milk preservative, especially in milk to be used by children, should be condemned. G. T. Welch (30) records some alarming instances of poisoning following the local application of large amounts of boracic acid; and Chittenden (31) observed that while borax in moderate amounts exerts no inhibitory action on the peptic and tryptic digestion of proteids, in larger quantities it retards the proteolytic activity of both of these digestive fluids. Later, Chittenden and Gies (32) made an exhaustive study of the action of borax and boric acid on nutrition, with especial reference to their effect on proteid metabolism, the experiments being made upon full-grown dogs. They found as the result of these studies that small doses of boric acid, up to 3 grams per diem, are practically without effect upon the proteid metabolism and the general nutrition of the animals, and that even moderate doses of borax are practically without effect. Large doses of borax tend to retard somewhat the assimilation of proteid and fatty foods, and with very large doses there is a tendency to diarrhea and an increased excretion of mucus. Borax and boric acid in very large amounts (equal to 1.5 to 2 per cent of the food) are liable to produce nausea and vomiting. Both borax and boric acid are quickly eliminated from the body, almost entirely through the urine, and in none of the experiments were any abnormalities in the urine observed.

Reference has already been made to the work of Rideal and Foulerton (20) on boric acid and formaldehyde as milk preservatives. In this connection it may be well to call attention again to their conclusions. According to these authors, (1) boric acid, 1:2,000, and formaldehyde, 1:50,000, are effective preservatives for milk for twenty-four hours; (2) in these quantities these substances have no appreciable effect on digestion or on the digestibility of foods thus preserved. On the other hand, according to F. J. Allen (quoted by Halliburton (25)), borax delays or prevents the rennin coagulation of milk. An excellent résumé of the earlier pharmacological work on boric acid and borax is given by Liebreich (33). We gather from the data which are there presented that since its introduction into medicine in the seventeenth and eighteenth centuries there have been occasional accidents and deaths from boric-acid poisoning. In these instances, which were comparatively rare, very large doses of boric acid and borax were employed, and in certain instances, at least, the bad results reached through the employment of these substances as drugs could be explained as resulting from a marked idiosyncrasy on the part of the patient, and in certain other instances, as pointed out by Liebreich, death and the bad effects following the use of these compounds were in all likelihood traceable to other causes. Among those who have observed bad effects following the administration of boric acid and borax may be mentioned Gowers, Evans, Molodenkow, Lemoine, Bruzelius, Warfwinge, Rasch, G. T. Welch, and others. (See Liebreich (33).) On the other hand, boric acid was early recognized as a mild antiseptic, and was recommended in surgery as a dressing for wounds by Lister, Godlee, and others. Particularly good results were obtained through its use by Cane, so that to-day the value of boric acid as a mild antiseptic wash and dressing powder is fully recognized and its use in these directions is extensive and far-reaching. It is concerning its effects on the system when taken internally, however, that the greatest differences of opinion prevail. Opposed to those who have described bad effects and even death following the administration of boric acid and borax, we have the testimony of other medical authorities regarding the harmless character of boric acid preparations. Liebreich (33) cites the cases described by Polli in Legendre's "Traite practique d'Antiseptique applique a la Therapeutique et l'Hygiene" of a soldier who swallowed 25 grams of boric acid without bad results. Polli cites the cases of eight persons who took 2 grams of boric acid in milk daily for forty-five days and 4 grams daily for twenty-three days without showing the slightest abnormal symptoms. Also the great Virchow, having observed his own urine to be abnormal, kept himself on an alkaline regimen for three months by the use of large doses of borax followed in the morning by Carlsbad water. The results reached are best given in his own words: "Ich fuhr 3 Monate lang mit meinem alkalischen Regime fort, und bis auf den heutigen Tag habe ich niemals weder Eiter abgesondert, noch Albumen, noch Cylinder producirt; mein Harn ist so klar wie der einer Jungfrau." Binswanger also conducted a series of tests upon himself with the view of determining the effect of boric acid. During one day he took 18 decigrams without effect, except possibly to increase his appetite. When he took two doses of 3.654 grams in two hours vomiting set in, and when he took the third dose later in the same day he again vomited, but after two hours regained his normal condition. G. T. Welch quotes Gaucher to the effect that the fatal dose of boracic acid is 2.5 ounces, continued for at least ten days. On the basis of these observations and also certain observations on himself and from results reached in his study of the effects of boric acid and borax on animals, such as dogs, rabbits, guinea pigs, etc., Liebreich (33) enters into a somewhat vigorous defense of the use of boric acid and borax as food preservatives. It is his opinion that much of the opposition to the use of boracic preparations for such purposes grows out of prejudices handed down from bygone times, and he calls attention to the fact that in this connection undue stress has been laid upon the accidents resulting from the use of boric acid in surgery, and that to a considerable extent the opposition to the use of boric acid and borax as food preservatives is founded upon conclusions drawn from imperfect experimental researches. To him the critical spirit of this later-day investigation of such subjects as food preservatives is a matter of regret, and in one of his communications, "Ueber Conservirung durch Borsaeure" (34), he inquires somewhat petulantly, "Who would have made the introduction of pickled meat, smoked beef, and such like dependent on a chemical or pharmacological investigation?" He emphasizes the fact that notwithstanding that borax and boric acid have been in use as food preservatives for a series of decades not a single case of injury to health has been observed. Lebbin (35) failed also to discover any harmful effect from eating meat preserved with boric acid, and hence points out that no objection can be urged against its use as a preservative. Tunnicliffe and Rosenheim (36) studied the influence of boric acid and borax on the general metabolism of three children, and arrived at the conclusion that small doses, up to 1 gram per day, continued for some time, exert no influence on the proteid metabolism in healthy or delicate children. Both boric acid and borax were quickly eliminated from the system, and neither substance affected the general health or well-being of the children in any way.

A second treatise by Liebreich (37) on the effect of boric acid and borax on the human system appeared in 1902, the object of which, according to the author, was to refute certain erroneous and insufficient observations likely to encourage prejudices against the use of these compounds. He criticises the observations of Robinson, Kister, Hanford, Röse, Rost, Rubner, Mattern, Heffte, Le Bon, and others, on the grounds that they are based on faulty and inaccurate observations; that the tests and observations are not decisive, that in certain instances they involve contradictions; that the boric acid and borax were not administered with food, but were taken directly into the system, and that in certain instances the real cause of the disturbance attributed to borax and boric acid was in all probability badly preserved meat; and by way of further confuting the results reached by other observers regarding the toxic action of boric acid and borax he cites the findings of Tunnicliffe and Rosenheim, to the effect that children increased in weight on a diet containing borax and boracic acid. Liebreich is of the opinion, therefore, that practical experience justifies the use of boric acid and borax as food preservatives. Wiley, Bigelow, and others (38), as the result of their study of the effect of boric acid and borax on man, have found that while the persons under experiment were in many instances made temporarily ill by the quantities of boric acid administered, at the end of the year, after the final after period, they appeared to be, and so expressed themselves, in better physical condition than when they entered on the experimental work.

It has already been pointed out that salicylic acid and benzoic acid are only rarely used as milk preservatives. This is fortunate, since both of these substances must be looked upon as toxic, to a degree at least, and the former, at least, seems to be more or less cumulative in its toxic effects upon the system. The injurious effects resulting from continuous small daily doses of salicylic acid were first pointed out by Brouardel (39), who made a plea for its discontinuance as a food preservative and for more thorough and systematic examinations of preserved foodstuffs by chemists and health officers. The effect of salicylic acid and the salicylates on man has also been investigated quite recently by Doctor Wiley (40) and his coworkers at the hygienic table. He points out in his general conclusions that there has been a general consensus of opinion among scientists and medical authorities that salicylic acid and its compounds are very harmful substances and that the prejudice against them is perhaps greater than against any other form of food preservatives. While he is still inclined to look upon it as a harmful substance, it is probably of less virulence than has heretofore been supposed.

Attention has already been called to the use of hydrogen peroxide in the sterilization of milk. In its 3 per cent solution this substance has been employed by Budde (16) to sterilize milk at somewhat lower temperatures than those employed in the ordinary processes of pasteurization, and attempts have also been made to remove all traces of the peroxide remaining in the milk after such treatment. According to Lakin (41), however, these attempts have not proven practicable, and this author therefore objects to Budde's process of sterilizing milk on the ground that it still contains small amounts of the unchanged hydrogen peroxide, and also in consequence of the injurious impurities which commercial solutions of hydrogen peroxide are liable to contain—such as boric acid and arsenic—which are present in the substances from which the solutions of hydrogen peroxide are made. He adds, however, that the consumption of milk sterilized by this method is not known to have produced any injurious effects. P. Gordan (42) has shown that the small amounts of hydrogen peroxide employed by Budde in his process of sterilizing milk have practically no sterilizing action, and that if employed in quantities sufficient to sterilize, it imparts a taste to the milk and renders it unfit for human consumption. According to a number of authorities hydrogen peroxide is apparently harmless in its effects. Amberg (43) quotes the following authorities on this point: Jablin-Gonnet (44) fed milk containing hydrogen peroxide to young animals and took it himself for two months without ill effect. Rosam (45) took within a period of three months a quantity of hydrogen peroxide, in milk, corresponding to 1,800 cubic centimeters of a 3 per cent solution without the least injurious effect, and Vandervelde (46) claims to have shown that hydrogen peroxide favors the action of rennin, pepsin, trypsin, and galactase.

Concerning the possibility of injurious effects resulting from the use of fluorides as milk preservatives, it may be said that the evidence now at hand goes to show that these substances are irritating poisons of considerable power. That such is the case may be seen from the following observations which have been made on their toxicity: Rubuteau (47) found that 0.5 gram of sodium fluoride given by the mouth produced sickness in dogs and 0.25 gram by mouth produced sickness in rabbits. When injected subcutaneously 0.25 gram of sodium fluoride proved fatal to rabbits. Kolipinski (48), who successfully employed sodium fluoride in minute doses in epilepsy, intermittent fever, and sympathetic headache, observed that 5 grains caused vomiting in a dog, when administered by the mouth, and that 3 grains injected into a dog or cat caused death in a few hours. The urine in such cases was found to contain small amounts of albumen, and to be rich in fluorine, indicating its elimination by the kidneys. Schulz (49) found the lethal dose of sodium fluoride for rabbits to be 0.2 to 0.4 gram, for dogs 0.3 gram, and for frogs 0.005 to 0.006 gram. Heidenhain (50) found the lethal dose for dogs to be 0.05 to 0.1 gram per kilo body weight. Weinland (51) observed that a 2.1 per cent solution of sodium fluoride killed the mucous membrane of the throat of a frog and Gruentzner (52) found that at such a concentration living nerves are destroyed. Czrellitzer (53) found it to be an active poison for all form of cells, and for protoplasm generally, but states that no satisfactory explanation of its toxicity is yet known. Kastle and Loevenhart (54) found it highly toxic to lipase, the fat-splitting ferment, and quite recently Loevenhart and Pierce (55) have considerably extended these observations, and have found that sodium fluoride retards the action of lipase when present in a solution of the ferment at the great dilution of one to one hundred million.

Baldwin (56) has called attention to several cases of accidental poisoning by sodium fluoride, that came under his observation, in which an insecticide consisting of sodium fluoride was mistaken for baking powder and used in the making of griddlecakes. In these cases violent vomiting and purging followed quickly after the eating of these cakes, which probably contained rather large amounts of the fluoride. These observations led the author to test the toxicity of sodium fluoride on himself. He found that 0.03 gram of sodium fluoride, eaten with bread, produced no effect. Neither did 0.09 gram taken a little later. 0.25 gram taken on an empty stomach produced nausea in two minutes, which effect reached its maximum in twenty minutes. During this time there was an increased flow of saliva and retching, but no vomiting. In about two hours these symptoms had subsided. Luncheon was then eaten, but without relish. Vomiting occurred immediately after eating, and slight nausea continued throughout the day on which the poison was taken. Baldwin concludes from his observations that sodium fluoride belongs to the class of less violent poisons, the characteristic symptoms being nausea, vomiting, and salivation.

According to Van Slyke (57) potassium dichromate is not a very violent poison, though not entirely harmless.

Concerning the physiological effects of such substances as common salt, sodium bicarbonate, etc., nothing need be said in this connection.

It is evident therefore that those who have made the closest study of the use of preservatives in food are very much divided in their opinion regarding the possibility of ill effects resulting from their use. Indeed the whole subject of food preservatives has been discussed from practically every standpoint. A priori, most of us would probably be inclined to proceed on the assumption that a substance which is toxic to micro-organisms is also toxic to the cells composing the tissues of man and the higher animals. In his testimony before the food-preservatives committee, London, Halliburton (25) took the stand that the use of food preservatives should be abandoned and methods of cold storage and transportation substituted in their place, upon the ground (1) that an antiseptic which is inimical to the life of those organisms that cause putrefaction can not be harmless to the vital processes of the higher animals; (2) numerous clinical observations have been recorded which show that dyspeptic and other troubles follow the use of foods which have been treated with preservatives ordinarily employed for such purposes, such as borax; (3) even if as in the case of boric acid and borax, the poison is not cumulative, the continuous passage of foreign substances through the kidneys can not be beneficial to those organs. A similar stand against the use of preservatives in food has been taken by Leffmann (58). According to this author, the bad effects of a food preservative may show itself in several ways: (1) It may interfere with the action of the digestive ferments, as has been proven in the case of salicylic acid; (2) it may act on the food, like formaldehyde; and (3) it may work a direct injury to the body as is known to be the case with almost all mineral preservatives. Hope (59) looks upon it as proven beyond dispute that chemical preservatives while checking the putrefactive changes in food, also check the fermentative processes of digestion. Especially does he regard the use of preservatives in milk as absolutely indefensible, and points out that the experiments of the bacteriological department of the Thompson-Yates laboratories are sufficient in themselves to establish the dangers of this practice, even if they stood alone. According to this author, there are numerous cases of injury resulting from the use of milk so preserved. He is therefore of the opinion that cleanliness and cold alone should be relied upon to insure the preservation of milk. Vaughan and Veenboer (60) have arrived at the conclusion that it is desirable to prevent the use of formaldehyde in any and all foods, and also not to allow the use of any preservatives in milk. They are of the opinion, however, that the use of one-fourth of 1 per cent of boric acid in cream would probably not prove harmful. The English commission appointed to inquire into the subject of food preservatives, upon the testimony and findings of seventy-eight experts, prohibited the use of all preservatives and coloring matters in milk, and at the International Congress of Hygiene, held at Brussels in 1903, resolutions were passed practically prohibiting the use of preservatives in all kinds of foods.

On the other hand, Rideal and Foulerton (61) contend that in view of the exceedingly perishable nature of milk, and the fact that it frequently has to be brought long distances before reaching the consumer, the use of a preservative is not only legitimate, but distinctly advantageous from a hygienic standpoint, providing that the preservative is not injurious to the health of the consumer. It may be said finally, however, that the preponderance of medical and scientific opinion is decidedly against the use of preservatives in milk, not only on account of possible injuries, especially to young children, resulting from the continued use of such preservatives in small amounts, but also for the reason that the use of such substances, if permitted, would ultimately tend to carelessness and uncleanliness in the handling of milk. Cleanliness and cold, the rigorous enforcement of the tuberculin test, and proper medical supervision of the dairies and those who handle the milk, are the prime essentials for a pure milk supply, and no method of sterilization or preservation is likely to give as good results.

In this connection, Richmond (62) has pointed out that in hot summer weather milk preservatives are comparatively useless unless added in relatively large quantities. He also calls attention to the fact that when once the souring of milk containing a small amount of preservative begins it proceeds at an increased rate as compared with milk to which no preservative has been added.

An actual case of milk adulteration which came under our observation at the Hygienic Laboratory will serve to illustrate the different phases of this subject. On July 23, 1907, a sample of milk was received from the Jamestown Exposition. According to the statement of the person submitting the sample, this milk was a sample of the milk supplied the guests at one of the tables of a hotel within the exposition grounds. This sample of milk gave the following numbers on analysis:

Specific gravity		1.0213
Fat	per cent	1.7
Total solids	do	7.5
Total solids not fat	do	5.80
Ash	do	.43
Milk sugar	do	3.37
Refractometer reading		32.1

It was also found to contain formaldehyde and to be artificially colored with an azo dye. It was also found to contain a large number of bacteria per cubic centimeter. The results of our examination of this milk show that the milk was watered. The fact that it contained a large number of micro-organisms despite the addition of formaldehyde indicates either that proper care had not been exercised in drawing the milk from the cow or that the proper care and cleanliness had not been exercised in handling it, or that the attempt had been made to keep it for too long a time and probably at too high a temperature. Such milk is not only below standard so far as food constituents is concerned, but it is exceedingly liable to infection, yet this was a sample of the milk probably supplied to many persons while they were guests at this hotel. This single instance is sufficient to illustrate the real significance of milk adulteration and its possible dangers.

PART V.—THE WASHINGTON MILK SUPPLY.

So far as our experimental work on this subject is concerned, the principal object has been to determine the general character of the milk at present supplied to the consumer in Washington and the District of Columbia. With this in view, routine chemical analyses have been made of milk offered for sale by various milk dealers in the city of Washington and the District of Columbia, from the 5th of July, 1907, to the 27th of September, 1907, inclusive. So much has been written on the subject of the routine analysis of milk, and the methods at present employed are generally so well understood, that only a few words concerning the methods employed in this investigation are required. For further details concerning methods of milk analysis, the reader is referred to the following standard works on this subject, viz, Modern Methods of Testing Milk and Milk Products, Van Slyke, New York, 1907; and Food Inspection and Analysis, Leach, New York, 1907. The chemical examination of the Washington milk supply has included the determination of specific gravity, total solids, fat, sugar, ash, acidity, refractometer reading, quantity of dirt by volume, and tests for preservatives. During the month of September special attention was paid to the examination for preservatives, and during this time the determination of sugar and total solids was omitted. The latter were calculated from the specific gravity and the percentage of fat according to Babcock's rule. The samples submitted for examination were collected by certain inspectors of the health office, and as soon as collected were put on ice and kept there until delivered at the Hygienic Laboratory, and until the chemical examinations were completed. As soon as the sample was brought into the laboratory, the acidity of the milk was determined on 50 cubic centimeters of the sample. The specific gravity and the percentage of fat and also the refractometer reading (the latter on the milk serum) were also determined practically as soon as the sample reached the laboratory, especially in those cases in which owing to lack of time the total solids were not determined by weighing, and in the event that these determinations indicated that any particular sample was below standard, the total solids on this particular sample were determined by weighing in the manner described in the following:

Specific gravity.—The specific gravity of the milk was determined either by means of the Westphal balance or by means of the Quevenne lactometer.

Total solids and ash.—The total solids and ash were determined by the method recommended by Leach (1). This method consists in heating 5 grams of the milk on the steam bath for three hours, in small flat platinum dishes. At the end of this time the dishes were removed from the steam bath and while still hot were wiped dry with a piece of soft toweling. They were then allowed to cool and weighed. In this way we obtained the weight of the residue from 5 grams of milk, and from this we calculated the percentage of total solids. The ash of the milk was then determined on the same sample by ignition at a low red heat, cooling and weighing the dish and its contents the second time. The ash left after this operation was tested for boric acid by the turmeric test.

Fat.—The quantity of butter fat in the milk was determined by the Babcock centrifugal method. This is the most rapid method known for the determination of fat in milk. It compares very favorably as to accuracy with the most exact methods now known for the determination of fat in milk, and it is the method ordinarily employed in practice for this purpose.

Lactose.—The amount of lactose in the several samples of milk was determined polarimetrically after the removal of the milk proteids by means of an acid solution of mercuric nitrate.

Acidity.—The acidity of the milk was determined by titrating 50 cubic centimeters of the sample with tenth-normal sodium hydroxide, using phenolphthalein as the indicator.

One cubic centimeter of tenth-normal sodium hydroxide, containing 0.004 gram of sodium hydroxide, is equivalent to 0.009 gram of lactic acid. Hence each cubic centimeter of tenth-normal sodium hydroxide required by the 50-cubic-centimeter sample of milk is equivalent to 0.018 per cent of lactic acid. In order, therefore, to obtain the per cent of acidity of the sample we multiply the number of cubic centimeters of tenth-normal sodium hydroxide required for neutralization by 0.018. The product is the acidity of the milk in percentage of lactic acid.

Thoerner (2) has suggested as a practical limit for wholesome milk an acidity equal to one-fifth of the volume of the milk in cubic centimeters of tenth-normal caustic soda. This would correspond to an acidity of 0.18 per cent of lactic acid. According to Van Slyke (3), the average acidity of English market milk, supposed to be 12 to 18 hours old, is 0.18 per cent, and of German milk 0.13 to 0.18 per cent. According to this author, market milk should not in any case contain over 0.2 per cent total acidity when it reaches the consumer, and generally should be under 0.15 per cent. According to Tuley (4), the milk of swill-fed cows is hyperacid.

Dirt.—The quantity of dirt or suspended matter in the milk may be estimated either gravimetrically (Renk, quoted by Ott (5)) or volumetrically (Van Slyke). The gravimetric method requires a large volume of milk and also requires considerable time. The volumetric method is rapid, and only small amounts of milk are required. For this reason the latter method was employed. Fifteen cubic centimeters of the sample was placed in a Bausch and Lomb graduated centrifuge tube. The samples were then centrifugalized for five minutes. The dirt then collects on the bottom of the tube, and the volume of it is read. From these readings the per cent of dirt is calculated.

Refractometer reading.—Milk serum has a higher index of refraction than water. Therefore the addition of water to milk lowers the index of refraction of the serum The refractometer reading of the several samples was obtained in the following manner: One hundred cubic centimeters of the sample of milk is placed in a beaker. To this 2 cubic centimeters of 25 per cent acetic acid is added. The beaker is then covered with a watch glass and heated in a water bath at 70° C. for twenty minutes. It is then placed in ice water for ten minutes and filtered. The refractometer reading on the clear yellowish filtrate (milk serum) is then made with a Zeiss immersion refractometer, at 20° C. A description of this instrument, together with all necessary directions for its use, is given by Leach (6). See also Wagner (7). Unadulterated milks give a refractometer reading varying between 39 and 43 on the scale of this instrument. According to Leach (8), a reading below 40 with the above conditions carefully observed would be suspicious of added water, though 39 might more safely be placed as a limit, below which milk could be declared fraudulently watered.

The following data given by Leach (9) show the variations in the specific gravity, refractometer reading, chemical composition, etc., resulting from the addition to a whole milk of various amounts of water up to 50 per cent.

Determinations on milk.							On milk serum.	
Added water.	Total solids.	Water.	Fat.	Solids not fat.	Ash.	Specific gravity at 15° C.	Specific gravity at 15° C.	Immersion refractometer reading at 20° C.
Per cent.	*Per cent.*	*Per cent.*	*Per cent.*	*Per cent.*	*Per cent.*			
0	12.65	87.35	4.00	8.65	0.65	1.0315	1.0287	42.40
10	11.33	88.67	3.50	7.83	.60	1.0278	1.0260	39.75
20	10.10	89.90	3.10	7.00	.53	1.0252	1.0230	36.90
30	8.95	91.05	2.80	6.15	.48	1.0211	1.0200	34.10
40	7.67	92.33	2.40	5.27	.40	1.0192	1.0167	31.10
50	6.43	93.57	2.00	4.43	.38	1.0154	1.0140	28.45

Coloring matters.—All of the samples of milk were examined systematically for artificial coloring matters by the methods given by Leach (10).

Preservatives.—All of the samples of milk were examined for antiseptics (1) by the souring test. That is, a portion of the sample was placed in a flask and allowed to stand overnight at room temper ature. If the milk turns sour in this time and curdles normally it was taken as an indication that antiseptics had probably not been added. On the other hand, if it did not curdle in this time, under these conditions, it was regarded as possibly containing preservatives and was systematically examined for all substances ordinarily employed as milk preservatives by the methods described by Leach, Van Slyke, and other well-known authorities on the subject.

(2) A considerable number of the samples were tested for preservatives by Blyth's (11) method for the detection and estimation of preservatives in milk. This method is carried out in the following manner: Ten cubic centimeters of the milk is put into a clean, wide test tube, and into another tube for purposes of comparison and control are put 10 cubic centimeters of a sample of milk of known purity. To each tube 2 cubic centimeters of a strong aqueous solution of blue litmus is then added, and after plugging with cotton wool the tubes are sterilized by heating to 80° C. for ten minutes. The tubes are then removed from the sterilizer and cooled to ordinary temperature. Each tube is then inoculated with 0.5 cubic centimeter of a solution containing 0.5 cubic centimeter of sour milk and 100 cubic centimeters of water. After thoroughly mixing, the tubes are kept at 15° to 25° C. for twenty-four hours and are then examined. The tubes containing samples of milk which contain preservatives will be colored blue or pink, whereas the tubes containing milks to which no preservatives have been added will be of the same color as the control experiment with normal milk, viz, white or nearly so. This test depends upon the fact that in the normal souring of milk the colored substances present in litmus are reduced by the bacteria to colorless (leuco) compounds.

(3) All of the samples of milk without exception were tested for formaldehyde and boric acid by the methods described by Leach (12). The test for formaldehyde described by this author as the hydrochloric acid test is capable of readily detecting 1 part of formaldehyde in 250,000 parts of sweet milk and 1 part in 50,000 in sour milk. It has been shown by Rideal and Foulerton (13) that at least 1 part of formaldehyde in 50,000 is required to preserve milk for twenty-four hours, so that this test is capable of detecting much smaller quantities of formaldehyde than is ever employed in practice. During the month of September, during which time special attention was paid to the subject of preservatives in the milk, 20 cubic centimeters of each sample of the milk was distilled and a few cubic centimeters of the distillate collected in a small amount of dis-

tilled water. The distillate was then tested for formaldehyde by a modification of the Hehner test (see Acree (14)), which in our hands enabled us to detect with certainty 1 part of formaldehyde in 1,000,000 parts of milk when a few drops of normal milk are used to supply the proteid required in this test.

In Table I are given the results of our analyses.

In Table II, column (1), are given the serial numbers of the samples of the milks of the several dairies. These are the inspectors' numbers furnished by the health office of the District of Columbia. In column (2) are given the total number of samples analyzed from each dairy. In column (3), the inspectors' numbers of such samples as were found to be below the standard of purity now fixed for the District of Columbia. In column (4), the total number of samples found to be below this standard. In column (5) are given the inspectors' numbers of the samples which were found to contain measurable amounts of dirt, viz, quantities equal to or greater than 0.07 per cent by volume of the milk. In column (6) are given the total number of milks from each dairy containing measurable amounts of dirt.

It will be seen from the totals given at the end of Table II that out of a total of 452 samples of milk analyzed 55 were found to be below standard, and of these which were found to be below standard 48 contained less than 3.5 per cent of fat and 17 gave evidence of having been watered. In addition to the 55 samples found to be below standard, 4 samples gave results indicating the probability of their having been watered, and 2 of the samples had probably been skimmed. It will also be seen from Table II that out of 452 samples analyzed 242 contained measurable amounts of dirt, varying from 0.07 per cent by volume of the milk to ten times this amount, viz, 0.7 per cent by volume. Only one of the samples out of the 452 analyzed was found to contain preservatives. This particular sample contained small amounts of boric acid. None of the samples contained artificial coloring matters. The following additional facts concerning certain of these samples are not without interest in this connection: Samples 148B, 235B, 240B, 241B, 280B, 297B, 1C, 2C, 8C, 44C, 58C, and 60C were put up in bottles containing stale milk. Samples 18A, 196B, 203B, 216B, and 237B were put up in dirty bottles. Feces were found in sample 110B, grass in sample 121B, pieces of straw in samples 51A, 57B, 114B, 151B, 169B, 215B, and 220B. Pieces of hair were found in samples 19A, 77B, 117B, 181B, and 198B. A blue substance, probably laundry bluing, was found in sample 155B;

and pieces of leaves in samples 121B and 196B; and one or more dead flies in samples 43B, 56B, and 252B.

Samples 28A, 4B, 29B, 51B, 91B, 110B, 132B, 154B, 179B, 189B, 199B, 208B, 234B, 247B, 248B, 255B, 277B, 21C, and 38C were found to contain more than 0.18 per cent of lactic acid.

Finally a word or two should be said as to the general import of these adulterations of the Washington milk supply. First, the fact that 48 of the samples analyzed contained less than 3.5 per cent of fat is not in itself a matter of serious import, for the reason that the milk of perfectly healthy cows frequently contains less than 3.5 per cent of fat, and yet no one could question the value of such milk as a food. Then again, we note that the requirements here in the District of Columbia regarding the fat content of milk are higher than the United States standard controlling the composition of milk offered for sale under the laws governing interstate commerce. This in itself may indicate possibly that the requirements governing the percentage of fat in milk within the District of Columbia are a trifle too high.

As already pointed out, the watering of milk is a practice which should be vigorously condemned and controlled by rigorous enforcement of the law, for the reason that such practice is not only fraudulent, but also a serious menace to the health of the community by reason of the fact that the milk may become infected with pathogenic organisms as the result of the addition of polluted water, and ordinarily the dairyman who waters his milk does not stop to consider the character of the water which he is adding thereto. In fact the degree of water pollution which might seriously contaminate and infect a milk supply, if the water were added to the milk, would probably under most circumstances be exceedingly difficult to detect. The only way therefore to control such a situation is simply to prevent by law the addition of water to milk in any form. According to Atlee (15), impure water is one of the most frequent sources of the pollution of milk, resulting either from the addition of water for purposes of adulteration or from its use for washing utensils. Winslow (16) is also of the opinion that water is probably the most dangerous adulterant of milk, for the reason that the water used by dairymen is frequently dirty and contaminated with pathogenic organisms.

From the standpoint of public health the point of chief interest and of the greatest importance brought out in this investigation is the large number of milks sold in Washington containing measurable amounts of dirt. Two hundred and forty-two samples out of 452, or 53.5 per cent of all the samples examined, contained 0.07 per cent, or more, of dirt by volume of the milk. Many more of the samples contained traces of dirt, and comparatively few were absolutely clean. During the summer of 1906, of 172 samples of milk examined in the Division of Pathology and Bacteriology of the Hygienic Laboratory, 98 samples were found to contain a very small amount of dirt. Eight contained much dirt, and 1 contained (mouse?) feces. (See Bulletin 35, Hygienic Laboratory, United States Public Health and Marine-Hospital Service, p. 71.) All sanitarians are agreed that milk should contain no dirt, and by the use of the Gurler milk pail in milking, and by taking a few simple precautions in the handling and preservation of milk it can certainly be kept out, and a good clean milk delivered to the consumer.

The presence of dirt in such a large percentage of the samples examined indicates an alarming neglect of even the simplest precautions, and probably accounts for the large number of bacteria found in the greater number of milks on sale in the city of Washington during the summer months. According to Renk (quoted by Ott (5)), cow's milk should be put on the market in such a state of purity that after two hours' standing a liter of the milk should show no appreciable deposit. Very few of the milks offered for sale in this city would conform to this requirement.

It should be observed in this connection, however, that dirty milk is by no means confined to this locality. Nearly every city throughout the world has to contend with this problem. According to some authorities, the citizens of Berlin consume 300 pounds of cow dung in their milk daily, and the citizens of New York consume 10 tons of filth and refuse in the same manner; and many medical authorities, among them Winslow (16), assert that the question of dirt and the bacterial contamination of milk is of infinitely greater importance from the standpoint of health than a high chemical standard governing the composition of milk, for the reason that very poor milk, viz, that which is low in proteids, fat, and milk sugar, is still very valuable as a food and contains a great deal of nutriment, provided that it is sufficiently clean to be consumed with safety. On the other hand, it is now perfectly well understood that dirty milk and milk bacterially contaminated is not only responsible for the high death rate prevailing among young children from cholera infantum, but that polluted milk is also responsible to a large degree for the spread of such infections as diphtheria, scarlet fever, typhoid fever, and tuberculosis, and for acute cases of milk poisoning, which are by no means uncommon.

It is therefore not surprising that some medical authorities (17) have gone so far as to express a preference for milk containing certain antiseptics, especially small amounts of formaldehyde, to the germ-laden milk ordinarily supplied the consumer in cities, and, for that matter, in many places in the country and even on the farm. The following communications on the subject of impure and dirty milk contain suggestions of great practical value:

"Impure milk and its evils," J. H. Atlee, Trans. Med. Soc. Tenn. 1897, 54-61.

"The clean-milk problem," Winslow, Northwest Medicine, Seattle, 1904, II, 315–327.

"Sources, effects, and prevention of dirty milk," Harrington, Amer. Jour. Pub. Hyg., 1904, 14, 31–55.

"Certified milk and the general milk supply of Louisville," Tuley, Jour. Amer. Med. Assn., 1907, 49, 1344–1349.

While it is more or less foreign to the general scope of this communication to discuss the economics of the milk question, it may not be amiss to point out that the production of clean, wholesome milk is largely a matter of cost and education. Medical authorities and practical dairymen and milk producers have alike, and more or less independently, arrived at the conclusion that clean, cold milk of a high grade of purity can not be sold to the consumer at less than from 8 to 10 cents a quart, and that in a number of instances where the production of such milk has been tried it had only a limited sale at 10 cents per quart, on account of the general apathy of even those persons well able to afford to pay this price. It is evident, therefore, that not only the dairyman, but also the general public, is in need of education regarding the necessity for a purer milk supply. It is also evident that a price of 8 to 10 cents a quart probably puts milk beyond the reach of the poorer classes. There are those who are of the opinion that pure high-grade milk can not be supplied to the poorer classes except by private philanthropy or municipal aid.

The fact that the Washington milk supply is practically free from preservatives and artificial coloring matters is one point in its favor. Thus considerable toward its purification and betterment has already been accomplished by the health officer of the District by the strict enforcement of the law regulating this subject. I understand that the results of the analyses of the Washington milk supply made in the health office and the Bureau of Chemistry, U. S. Department of Agriculture, practically confirm the results reached in the Division of Chemistry of the Hygienic Laboratory regarding the freedom of the milk from preservatives and artificial coloring matters.

TABLE I.—*Analyses of milk sold in Washington and the District of Columbia.*

[Hygienic Laboratory, Division of Chemistry, July 5, 1907, to September 28, 1907, inclusive.]

No. of sample.	Date.	Specific gravity.	Fat.	Total solids.	Solids not fat.	Ash.	Milk sugar.	Acidity.	Sediment.	Refractometer reading.
	1907.									
1A	July 5	1.0288	4.0	14.11	10.11	0.58				
2A	do	1.0335	3.8	13.89	10.09	.62				
3A	do	1.0315	3.6	11.83	8.23	.637				
4A	do	1.0308	3.5	12.50	9.0	.62				
5A	do	1.0298	4.0	12.94	8.94	.60				
6A	do	1.0327	3.3	12.72	9.42	.66				
7A	July 6	1.030	4.4	14.20	9.8	.65			(a)	
8A	do	1.031	4.25	13.72	9.47	.62			(a)	
9A	do	1.0256	4.3	12.88	8.58	.70			(b)	
10A	do	1.0313	4.0	13.41	9.41	.81			None.	
11A	do	1.030	4.0	12.49	8.49	.64			(b)	
12A	July 8	1.029	3.7	12.05	8.35	.61	4.51		(b)	41.3
13A	do	1.032	3.9	8.97	5.07	.68	4.90		None.	40.5
14A	do	1.029	3.8	14.87	11.07	.67	4.56		(b)	39.5
15A	do	1.033	3.8	13.59	9.79	.60	4.99		(a)	42.5
16A	do	1.031	2.8	11.29	8.49	.64	4.71		(a)	41.0
17A	July 9	1.031	3.8	12.54	8.74	.71	4.68	0.133	None.	42.2
18A	do	1.032	3.15	12.30	9.15	.70	4.57	.141	(c)	41.5
19A	do	1.032	4.8	14.10	9.30	.71	4.06	.153	(c)	43.0
20A	do	1.031	4.4	13.47	9.07	.65	4.93	.162	(b)	41.7
21A	do	1.0315	3.8	12.40	8.6	.70	4.70	.155	(a)	41.25
22A	July 10	1.030	4.2	15.13	10.93	.71	4.76	.134	(b)	41.3
23A	do	1.031	4.8	13.87	9.07	.68	4.59	.137	(a)	41.5
24A	do	1.0257	3.1	10.62	7.52	.57	3.69	.122	(c)	36.5
25A	do	1.030	4.0	12.68	8.68	.65	4.67	.141	(a)	41.5
26A	do	1.031	3.8	12.35	8.55	.63	4.69	.130	None.	41.5
27A	July 11	1.0312	4.4	13.34	8.94	.73	4.55	.146	(b)	42.4
28A	do	1.0294	5.0	13.83	8.83	.65	4.61	.185	(a)	42.25
29A	do	1.031	5.0	14.35	9.35	.65	4.90	.158	(b)	43.2
30A	do	1.0328	4.6	14.00	9.40	.68	4.83	.150	(c)	43.5
31A	do	1.031	3.2	12.31	9.11	.66	4.63	.141	None.	42.1
32A	July 12	1.033	3.6	13.00	9.4	.75	4.91	.157	(c)	43.5
33A	do	1.029	5.0	13.30	8.30	.68	3.97	.135	(a)	40.0
34A	do	1.031	4.95	12.23	7.28	.63	4.46	.137	None.	41.2
35A	do	1.030	3.8	12.60	8.8	.60	4.46	.137	(c)	41.0
36A	do	1.034	3.5	12.49	8.99	.67	5.05	.140	(b)	42.5
37A	do	1.031	3.7	12.92	9.22	.67	4.90	.157	(c)	41.2
38A	do	1.029	4.8	12.48	7.68	.63	4.74	.130	None.	40.5
39A	do	1.0284	4.4	13.52	9.12	.70	4.85	.131	(b)	41.6
40A	July 13	1.030	3.8	12.83	9.03	.67	4.76	.135	0.13	40.5
41A	do	1.033	3.0	12.55	9.55	.59	4.85	.141	.20	41.2
42A	do	1.030	3.7	12.59	8.89	.58	4.65	.141	Trace.	40.0
43A	do	1.032	4.2	13.24	9.04	.62	4.71	.142	.07	41.3
44A	do	1.033	4.6	13.64	9.04	.55	4.91	.178	.07	42.5
45A	July 15	1.031	4.6	13.78	9.18	.67	4.62	.144	Trace.	42.25
46A	do	1.033	4.6	14.37	9.77	.72	4.90	.137	Trace.	43.0
47A	July 15	1.033	4.4	13.76	9.36	.65	4.87	.158	.33	40.5
48A [d]	do	1.030	3.1	11.73	8.63	.60	4.69	.142	.13	44.0
49A [e]	do	1.033	3.8	13.0	9.2	.67	4.93	.149	.06	42.0

[a] Slight. [b] Very slight. [c] Considerable. [d] Full of dirt. [e] Contained hair.

TABLE I.—*Analyses of milk sold in Washington and the District of Columbia*—Cont'd.

No. of sample.	Date.	Specific gravity.	Fat.	Total solids.	Solids not fat.	Ash.	Milk sugar.	Acidity.	Sediment.	Refractometer reading.
	1907.									
50A	July 15	1.032	3.0	11.65	8.65	.61	4.77	.139	.13	42.0
51A[a]	do	1.032	4.0	13.44	9.44	.66	5.40	.148	.13	43.0
52A	July 16	1.030	3.4	12.31	8.91	.71	4.78	.142	Trace.	41.25
53A	do	1.031	4.0	12.89	8.89	.69	4.61	.139	Trace.	41.0
54A	do	1.032	3.8	13.06	9.26	.69	4.96	.166	.13	43.0
55A[b]	do	1.031	3.1	12.26	9.16	.70	4.46	.137	.20	42.0
56A	do	1.031	4.2	13.14	8.94	.67	4.73	.139	.07	42.0
57A	do	1.029	2.4	11.81	9.41	.61	4.44	.128	.27	39.0
58A	do	1.033	4.6	14.27	9.67	.67	4.81	.139	Trace.	42.5
59A	July 17	1.0285	6.6	12.89	6.29	.74	4.66	.15	.07	42.0
60A	do	1.0303	4.2	13.28	9.08	.73	4.55	.140	Trace.	40.5
61A	do	1.0322	4.2	13.80	9.60	.73	4.81	.121	Trace.	41.4
62A	do	1.0312	3.8	13.12	9.32	.73	4.50	.138	Trace.	41.0
63A	do	1.0312	4.6	12.49	7.89	.72	4.28	.137	Trace.	42.0
64A	do	1.0281	4.0	11.86	7.86	.62	4.12	.139	.33	38.5
65A	do	1.0323	4.6	14.20	9.60	.71	4.90	.142	.20	43.0
66A	do	1.0303	5.6	14.15	8.55	.68	4.74	.135	.13	41.5
67A	July 18	1.0289	4.1	12.37	8.27	.66	4.30	.139	Trace.	40.1
68A	do	1.029	3.4	11.59	8.19	.67	4.51	.130	Trace.	39.4
69A	do	1.0326	4.8	15.06	10.26	.71	4.29	.158	.07	43.5
70A	do	1.0275	5.4	13.13	7.73	.63	4.31	.126	.20	39.0
71A	do	1.0315	4.2	13.24	9.04	.67	4.75	.146	.13	42.2
72A	do	1.0297	5.8	14.49	8.69	.66	4.78	.149	.20	41.2
73A	July 19	1.0324	5.0	14.07	9.07	.75	4.56	.144	.07	42.0
74A	do	1.0315	3.6	12.34	8.74	.67	4.75	.137	Trace.	41.0
75A	do	1.0312	4.3	13.55	9.25	.69	4.61	.140	.20	41.1
76A	do	1.0323	4.0	13.16	9.16	.71	4.94	.135	.33	42.0
77A	do	1.0314	3.7	12.68	8.98	.66	4.73	.142	.33	40.5
78A	do	1.0263	3.0	10.12	7.12	.49	3.77	.177	.07	36.5
79A	July 22	1.0315	3.2	12.77	9.57	.71	4.77	.135	.07	41.3
80A	do	1.0319	4.1	13.02	8.92	.67	4.48	.137	.26	41.0
81A	do	1.0309	4.6	13.69	9.09	.65	4.92	.157	Trace.	41.7
82A	do	1.032	3.4	12.21	8.81	.70	4.94	.137	.13	41.5
83A	do	1.0324	4.3	13.36	9.06	.67	4.93	.142	Trace.	42.0
84A	do	1.0313	4.2	12.61	8.41	.60	4.63	.146	Trace.	42.5
85A	do	1.0228	9.2	16.85	7.65	.52	4.22	.140	.33	39.5
1B	July 23	1.0314	3.6	11.80	8.2	.65	4.59	.137	Trace.	41.0
2B	do	1.032	5.6	14.27	8.67	.65	4.94	.146	Trace.	43.0
3B	do	1.0278	3.5	12.81	9.31	.62	4.18	.121	.07	38.5
4B	do	1.0319	4.6	12.95	8.35	.68	4.63	.216	.07	41.5
5B	do	1.0315	5.4	13.59	8.19	.67	4.63	.153	Trace.	42.0
6B	do	1.024	8.0	14.08	6.08	.58	4.14	.119	Trace.	37.8
7B	do	1.029	5.4	12.83	7.43	.63	4.68	.169	.13	40.5
8B	July 24	1.0301	4.5	13.21	8.71	.66	4.96	.130	Trace.	41.5
9B	do	1.030	5.8	14.50	8.70	.66	4.91	.139	None.	41.5
10B	do	1.0327	3.9	12.60	8.70	.67	5.00	.139	None.	42.5
11B	do	1.032	4.8	13.85	9.05	.60	4.71	.162	Trace.	42.0
12B	do	1.0315	3.8	12.73	8.93	.658	4.79	.139	.13	41.7
13B	do	1.0305	4.4	12.75	8.35	.67	4.78	.133	.07	41.0
14B	do	1.0315	3.8	12.59	8.79	.67	4.48	.137	.07	41.5
15B	July 24	1.0315	4.3	12.84	8.54	.67	4.75	.131	Trace.	41.1
16B	July 25	1.032	4.4	12.79	8.39	.66	4.69	.140	.33	42.0

[a] Pieces of straw.

[b] Large black particles.

TABLE I.—*Analyses of milk sold in Washington and the District of Columbia*—Cont'd.

No. of sample.	Date.	Specific gravity.	Fat.	Total solids.	Solids not fat.	Ash.	Milk sugar.	Acidity.	Sediment.	Refractometer reading.
	1907.									
17B	July 25	1.0303	7.2	15.44	8.24	.69	4.53	.151	.13	41.5
18B	do	1.0313	4.8	12.96	8.16	.66	4.90	.138	Trace.	42.0
19B	do	1.0324	5.0	13.92	8.92	.67	5.04	.155	.13	42.5
20B	do	1.0285	7.6	15.00	7.40	.59	4.82	.133	Trace.	42.1
21B	do	1.0329	5.4	13.78	8.38	.68	4.87	.137	.07	41.0
22B	do	1.0309	4.8	12.71	7.91	.71	4.69	.159	None.	41.2
23B	do	1.034	4.6	12.90	8.30	.67	5.09	.157	.33	43.0
24B	July 26	1.0316	3.4	12.84	9.44	.70	5.04	.148	.07	42.0
25B	do	1.0316	3.5	12.13	8.63	.68	4.88	.137	.07	41.0
26B	do	1.0277	3.8	11.61	7.81	.58	4.20	.119	.20	39.0
27B	do	1.0317	4.8	13.82	9.02	.67	4.92	.153	Trace.	40.0
28B	do	1.0299	3.5	11.67	8.17	.66	4.49	.155	.07	40.0
29B	do	1.0278	4.8	12.30	7.50	.48	4.20	.241	Trace.	39.0
30B	do	1.030	3.4	11.63	8.23	.61	4.43	.146	Trace.	40.0
32B	July 29	1.0309	5.7	15.68	9.98	.69	4.86	.133	.33	41.5
33B	do	1.0318	3.4	12.40	9.00	.70	4.52	.155	.20	41.5
34B	do	1.0323	4.3	14.07	9.77	.73	4.90	.157	.13	42.0
35B	do	1.0314	3.5	12.43	8.93	.68	4.63	.150	.26	41.0
36B	do	1.0316	4.2	13.79	9.59	.74	4.94	.148	.13	42.5
37B	do	1.0326	3.4	12.78	9.38	.72	5.00	.158	.26	42.2
38B	do	1.0315	4.0	13.20	9.20	.72	4.59	.173	.20	41.5
39B	do	1.0327	3.8	13.15	9.35	.71	5.06	.158	Trace.	42.0
40B	July 30	1.033	5.0	15.51	10.51	.70	4.85	.145	.07	42.2
41B	do	1.0316	4.8	14.67	9.87	.63	5.00	.144	Trace.	41.5
42B	do	1.0312	4.0	13.40	9.40	.67	4.66	.167	.07	40.5
43B[a]	do	1.0301	4.8	14.73	9.93	.65	5.11	.153	Trace.	42.0
44B	do	1.0301	3.5	13.09	9.59	.68	4.55	.133	.70	41.5
46B	do	1.0305	4.4	13.06	8.66	.66	4.44	.150	.33	41.0
49B	July 31	1.0323	4.6	14.38	9.78	.68	5.06	.166	.13	42.0
50B	do	1.0306	5.0	13.91	8.91	.68	4.86	.141	.07	41.0
51B	do	1.0325	3.5	12.59	9.09	.67	4.83	.184	Trace.	40.0
52B	do	1.0306	4.0	12.78	8.78	.70	5.03	.169	.07	40.3
53B	do	1.0316	3.8	13.90	10.10	.67	4.90	.155	Trace.	40.7
54B	Aug. 1	1.0288	4.2	12.40	8.20	.57	4.51	.146	.07	39.0
55B	do	1.0306	4.8	13.33	8.53	.61	4.74	.131	.07	40.2
56B[b]	do	1.0288	3.1	10.81	7.71	.54	4.77	.139	Trace.	38.3
57B[c]	do	1.0298	4.4	12.85	8.45	.63	4.72	.131	.20	40.5
58B	do	1.0279	5.5	12.82	7.32	.63	4.24	.142	Trace.	39.1
59B	do	1.0315	3.8	12.50	8.70	.62	4.81	.131	.13	41.0
60B	do	1.0305	5.3	13.60	8.30	.66	4.77	.140	None.	41.0
61B	do	1.030	3.1	11.36	8.26	.61	4.60	.133		39.3
62B	Aug. 2	1.0339	4.0	13.29	9.29	.72	4.91	.176	Trace.	42.0
63B	do	1.0316	4.7	13.14	8.44	.69	4.91	.146	.07	41.0
64B	do	1.0305	3.8	12.24	8.44	.67	4.70	.144	.13	40.1
65B	do	1.0306	2.6	10.77	8.17	.64	4.47	.158		39.0
66B	do	1.0288	3.6	11.03	7.43	.58	4.39	.130	None.	39.0
67B	do	1.0317	4.4	13.01	8.61	.65	4.81	.148	.07	41.0
68B	do	1.031	4.0	13.57	9.57	.67	4.72	.146	None.	41.2
69B	do	1.031	4.6	13.03	8.43	.69	4.70	.149	Trace.	41.5
70B	Aug. 5	1.0317	3.3	12.61	9.31	.70	4.93	.157	.07	41.5
71B	do	1.0315	4.5	13.31	8.81	.69	4.72	.144	.13	42.0
72B	do	1.0325	4.4	13.28	8.88	.70	4.87	.149	Trace.	41.5

[a] Two dead flies.

[b] Dead fly.

[c] Pieces of straw.

TABLE I.—*Analyses of milk sold in Washington and the District of Columbia*—Cont'd.

No. of sample.	Date.	Specific gravity.	Fat.	Total solids.	Solids not fat.	Ash.	Milk sugar.	Acidity.	Sediment.	Refractometer reading.
	1907.									
73B	Aug. 5	1.0316	4.0	12.71	8.71	.68	4.74	.155	.07	41.0
74B	do	1.0327	3.4	12.38	8.98	.72	4.51	.144	Trace.	41.2
75B	do	1.0309	4.2	12.74	8.54	.63	4.74	.128	Trace.	41.0
76B	do	1.031	5.5	14.05	8.55	.69	4.70	.137	.07	41.0
77B[a]	do	1.0319	3.6	12.52	8.92	.64	4.77	.164	Trace.	41.5
78B	Aug. 6	1.0313	3.8	13.43	9.63	.75	5.12	.167	None.	41.0
79B	do	1.030	4.2	11.92	7.72	.61	4.85	.133	.07	39.0
80B	do	1.0309	5.5	14.39	8.89	.64	5.05	.157	.13	42.0
81B	do	1.0269	4.8	11.84	7.04	.54	4.20	.117	.13	39.0
82B	do	1.0305	3.6	12.20	8.60	.62	4.60	.142	.20	40.0
83B	do	1.0289	4.0	12.05	8.05	.58	4.32	.146	.07	39.0
84B	do	1.0269	3.0	10.25	7.25	.53	3.95	.130	.13	36.5
85B	do	1.031	5.2	13.62	8.42	.73	4.18	.131	.07	41.0
86B	Aug. 7	1.0325	4.7	13.54	8.84	.64	4.79	.160	.13	41.5
87B	do	1.0327	5.9	15.31	9.41	.64	5.08	.166	.07	43.0
88B	do	1.0308	3.7	12.30	8.60	.63	4.81	.150	.07	40.3
89B	do	1.032	4.2	13.00	8.80	.63	4.77	.158	Trace.	41.2
90B	do	1.0325	3.8	12.83	9.03	.61	4.87	.167	Trace.	42.0
91B	do	1.031	5.3	13.67	8.37	.68	4.49	.205	.13	41.2
92B	do	1.032	4.3	12.88	8.58	.65	4.64	.167	.07	41.5
93B	do	1.031	4.0	12.45	8.45	.73	4.81	.135	Trace.	41.0
94B	Aug. 8	1.0313	4.6	13.48	8.88	.70	4.66	.149	.07	40.5
95B	do	1.0291	4.8	13.21	8.41	.58	4.49	.137	Trace.	38.5
96B	do	1.0322	4.6	13.64	9.04	.65	5.01	.159	.13	41.0
97B	do	1.0332	4.2	13.42	9.22	.65	5.18	.153	Trace.	42.0
98B	do	1.0313	4.3	12.91	8.61	.65	4.83	.137	Trace.	41.0
99B	do	1.0335	3.4	12.30	8.90	.68	4.77	.151	.07	42.0
100B	do	1.0305	4.5	13.05	8.55	.69	4.70	.151	.13	41.0
101B	do	1.0326	4.4	13.04	8.64	.65	5.10	.149	.20	42.0
102B	Aug. 9	1.0332	4.4	13.49	9.09	.68	4.87	.153	Trace.	42.5
103B	do	1.0308	4.2	12.67	8.47	.61	4.43	.142	.07	40.0
104B	do	1.0319	4.2	13.20	9.00	.62	4.32	.149	.07	40.5
105B	do	1.0315	4.1	13.12	9.02	.70	4.68	.144	Trace.	41.0
106B	do	1.031	3.4	12.09	8.69	.65	4.74	.155	.07	41.0
107B	do	1.031	4.6	13.39	8.79	.63	4.74	.148	None.	41.0
108B	do	1.0315	3.4	12.95	9.55	.64	4.60	.158	.07	41.1
109B	do	1.0301	4.3	11.59	7.29	.66	4.28	.130	.07	40.0
110B[b]	Aug. 12	1.0303	3.8	11.79	7.99	.62	4.79	.230	Trace.	40.0
111B	do	1.0314	3.8	12.43	8.63	.66	4.83	.157	.07	41.0
112B	do	1.0314	3.6	12.44	8.84	.62	4.79	.149	.07	40.0
113B	do	1.0314	7.0	14.92	7.92	.71	4.62	.139	.33	41.0
114B	do	1.0253	5.9	14.06	8.16	.59	4.20	.131	.20	38.0
115B	do	1.0329	4.4	13.29	8.89	.67	4.77	.148	.07	41.5
116B	do	1.0317	3.6	12.14	8.54	.61	4.70	.158	......	41.0
117B	do	1.0327	4.2	13.41	9.21	.51	5.16	.167	.33	42.0
119B	Aug. 13	1.0271	2.9	10.46	7.56	.64	3.97	.129	.07	36.5
120B	do	1.032	4.0	12.99	8.99	.69	4.91	.151	Trace.	40.1
121B[c]	do	1.031	4.1	12.58	8.48	.70	4.74	.139	None.	39.5
122B	do	1.033	4.6	13.74	9.14	.73	4.79	.146	None.	42.0
123B	do	1.030	4.0	12.71	8.71	.72	4.54	.146	.20	41.5
124B	do	1.0312	3.8	13.13	9.33	.75	4.74	.144	.13	41.0
126B	do	1.0301	4.2	12.88	8.68	.71	4.99	.133	None.	41.0

[a] Hair. [b] Contained feces. [c] Contained grass.

TABLE I.—*Analyses of milk sold in Washington and the District of Columbia*—Cont'd.

No. of sample.	Date.	Specific gravity.	Fat.	Total solids.	Solids not fat.	Ash.	Milk sugar.	Acidity.	Sediment.	Refractometer reading.
	1907.									
127B	Aug. 14	1.0298	4.2	12.67	8.47	.65	4.64	.158	.13	41.0
128B	....do	1.0314	4.0	13.04	9.04	.70	4.87	.162	.33	41.5
129B	....do	1.0303	5.0	14.07	9.07	.64	4.87	.140	Trace.	41.0
130B	....do	1.030	3.5	12.10	8.60	.66	4.64	.149	.07	40.0
131B	....do	1.0294	4.8	13.36	8.56	.63	4.81	.158	.27	41.0
132B	....do	1.0285	6.8	14.83	8.03	.62	4.22	.204	.13	42.0
133B	Aug. 14	1.0332	4.0	13.39	9.39	.69	4.66	.144	Trace.	43.0
134B	....do	1.0215	19.0	21.79	2.79	.57	4.77	.157	.33	42.5
135B	Aug. 15	1.0308	3.6	12.03	8.43	.68	4.70	.144	.13	40.0
136B	....do	1.0285	3.6	12.18	8.58	.69	4.62	.166	.07	40.2
137B	....do	1.0314	4.4	12.24	7.84	.62	4.39	.122	.20	38.5
138B	....do	1.0296	2.6	10.30	7.70	.66	4.64	.140	....	38.0
139B	....do	1.0315	3.5	12.27	8.77	.71	4.60	.140	.33	40.0
140B	....do	1.0325	4.5	13.27	8.77	.72	4.87	.160	....	42.0
141B	....do	1.0312	4.4	13.06	8.63	.67	4.74	.171	....	41.8
142B	...do	1.0298	3.6	11.69	8.09	.62	4.74	.142	....	40.0
144B [a]	Aug. 16	1.0271	8.7	16.59	7.89	.60	4.41	.173	.13	41.0
145B	....do	1.0327	4.4	13.67	9.27	.62	5.23	.166	None.	42.0
146B	....do	1.0327	5.4	14.62	9.22	.66	5.23	.142	.26	42.0
147B [b]	....do	1.0339	4.0	13.44	9.44	.62	5.18	.166	.13	42.2
148B [c]	....do	1.0278	3.6	11.65	8.05	.55	4.74	.131	Trace.	41.5
149B	....do	1.0308	4.2	12.73	8.53	.55	4.91	.142	Trace.	39.0
150B	....do	1.0298	4.4	12.78	8.38	.51	4.64	.146	.13	39.0
152B	....do	1.0299	4.7	12.72	8.02	.54	4.72	.149	.07	39.2
153B	Aug. 19	1.0305	3.8	12.51	8.71	.65	4.83	.142	.07	41.2
154B [a]	....do	1.0293	4.2	12.40	8.20	.654	4.54	.248	.13	40.5
155B [d]	....do	1.0314	3.6	12.48	8.88	.66	4.74	.144	.13	40.3
156B	....do	1.0294	3.6	11.88	8.28	.60	4.66	.142	.07	40.0
157B	....do	1.0305	3.8	12.24	8.44	.716	4.72	.133	Trace.	39.0
158B	....do	1.0286	3.8	11.31	7.51	.57	4.41	.135	.07	40.0
159B	....do	1.0315	3.6	12.426	8.826	.67	4.72	.148	.13	41.0
160B	....do	1.0315	3.6	12.45	8.85	.638	4.91	.151	.07	42.0
161B	Aug. 20	1.032	3.7	12.46	8.76	.66	5.08	.153	.07	41.0
162B	....do	1.032	4.6	13.66	9.06	.70	4.95	.144	.20	41.2
163B	....do	1.033	4.4	13.31	8.91	.694	5.08	.157	.07	42.0
164B	....do	1.0315	4.2	13.12	8.92	.714	4.97	.149	Trace.	41.5
165B	....do	1.032	3.8	12.926	9.126	.716	4.89	.153	.53	41.0
166B	....do	1.033	4.6	13.54	8.94	.66	5.14	.135	.07	42.0
167B	....do	1.031	4.2	12.71	8.51	.69	4.87	.131	.07	40.8
168B	....do	1.030	5.3	13.67	8.37	.69	4.93	.140	None.	41.0
169B [a]	Aug. 21	1.0299	4.0	12.70	8.70	.73	4.45	.139	.20	40.0
171B	....do	1.031	4.1	13.15	9.05	.71	4.97	.140	.07	41.0
172B	....do	1.0325	3.8	13.04	9.24	.66	5.16	.149	.13	41.0
173B	....do	1.031	3.0	12.07	9.07	.67	4.70	.139	.40	40.2
174B	....do	1.031	3.6	12.70	9.10	.70	5.03	.151	.07	40.6
175B	....do	1.0308	3.8	12.58	8.78	.72	4.77	.139	Trace.	41.0
176B	....do	1.0268	5.3	13.66	8.36	.53	4.49	.155	Trace.	39.0
178B	Aug. 22	1.031	6.1	14.88	8.78	.65	5.01	.133	.07	43.0
179B	....do	1.035	4.8	14.27	9.47	.69	5.33	.190	Trace.	42.3
180B	....do	1.0306	4.7	13.11	8.41	.63	4.91	.133	Trace.	41.5
181B [b]	....do	1.0316	4.2	12.83	8.63	.68	5.12	.173	Trace.	42.2

[a] Contained straw. [b] Contained hair. [c] Stale milk in bottle. [d] Blue substance in bottle.

TABLE I.—*Analyses of milk sold in Washington and the District of Columbia*—Cont'd.

No. of sample.	Date.	Specific gravity.	Fat.	Total solids.	Solids not fat.	Ash.	Milk sugar.	Acidity.	Sediment.	Refractometer reading.
	1907.									
182B...	Aug. 22	1.0302	5.4	14.13	8.73	.66	4.87	.158	.13	42.0
183B...	....do...	1.0308	4.3	12.474	8.174	.654	4.62	.142	.07	40.5
184B...	....do...	1.0321	4.2	12.90	8.70	.69	4.74	.148	.07	40.8
185B...	....do...	1.0312	3.8	12.44	8.64	.69	4.81	.148	.13	41.0
186B...	Aug. 23	1.0336	4.0	13.088	9.088	.72	4.99	.153	Trace.	42.0
187B...	....do...	1.0298	4.2	12.658	8.458	.67	4.70	.142	.07	40.1
188B...	....do...	1.0313	4.0	12.76	8.76	.69	4.66	.149	.07	40.2
189B...	....do...	1.0309	3.8	12.16	8.36	.69	4.70	.198	.13	40.5
190B...	....do...	1.032	4.8	13.634	8.834	.77	4.91	.146	.13	40.5
191B...	....do...	1.0306	4.8	12.96	8.16	.70	4.22	.106	.26	42.0
192B [a]..	Aug. 23	1.0255	6.0	12.38	6.38	.60	3.91	.130	.07	38.0
193B...	....do...	1.0321	4.2	12.90	8.70	.67	4.83	.149	....	41.0
194B...	Aug. 26	1.0294	4.6	12.54	7.94	.67	....	.135	.26	39.0
195B...	....do...	1.0325	4.4	13.33	8.93	.74	....	.149	.33	40.0
196B [b]..	....do...	1.035	5.7	14.37	8.67	.70	....	.160	Trace.	41.0
197B...	....do...	1.0326	4.0	12.91	8.91	.72	....	.151	.07	41.0
198B [c]..	....do...	1.0318	5.0	14.03	9.03	.70	....	.135	.08	41.2
199B...	....do...	1.033	4.2	13.59	9.39	.69	....	.189	Trace.	42.0
200B...	....do...	1.031	5.6	14.26	8.66	.57	....	.165	None.	41.8
201B...	....do...	1.0314	4.0	12.68	8.68	.68	....	.140	.20	41.5
202B...	Aug. 27	1.0305	4.0	12.63	8.63	.70	....	.156	.07	40.2
203B [d]..	....do...	1.0287	3.8	11.67	7.87	.66	....	.120	.33	40.0
204B...	....do...	1.0315	4.4	13.17	8.77	.66	....	.144	.07	42.0
205B...	....do...	1.0325	3.9	13.15	9.25	.71	....	.171	.13	42.5
206B...	....do...	1.0305	4.0	12.54	8.54	.71	....	.140	.07	40.5
207B...	....do...	1.0305	4.0	12.73	8.73	.61	....	.151	.13	41.0
208B...	....do...	1.0317	5.0	14.04	9.04	.76	....	.182	Trace.	42.0
209B...	....do...	1.0291	3.8	12.01	8.21	.67	....	.149	.20	41.0
210B...	Aug. 28	1.0294	6.3	13.94	7.64	.79	....	.157	None.	40.5
211B...	....do...	1.0308	5.0	12.75	7.75	.69	....	.153	.13	40.1
212B...	....do...	1.0283	4.6	11.78	7.18	.59	....	.137	Trace.	39.0
213B...	....do...	1.0314	6.0	13.99	7.99	.68	....	.144	None.	41.0
214B...	....do...	1.0319	4.6	13.00	8.40	.71	....	.146	.07	41.0
215B [e]..	....do...	1.0304	5.2	12.99	7.79	.71	....	.148	.13	40.0
216B [d]..	....do...	1.0314	5.6	13.56	7.96	.76	....	.155	.13	41.0
217B...	....do...	1.0286	4.3	11.73	7.43	.63	....	.155	....	39.1
218B...	Aug. 29	1.031	4.5	13.35	8.85	....	....	.149	.07	41.5
219B...	....do...	1.031	3.4	11.92	8.52	....	....	.142	.07	40.5
220B [f]..	....do...	1.0325	3.5	12.31	8.81	.73	....	.149	.07	41.5
221B...	....do...	1.0307	4.0	12.20	8.20	.69	....	.124	Trace.	41.0
222B...	....do...	1.0295	3.2	11.19	7.99	.66	....	.126	.07	39.2
223B...	....do...	1.0309	4.0	12.72	8.72	.69	....	.149	.07	41.0
224B...	....do...	1.0308	4.6	13.49	8.89	.65	....	.157	Trace.	41.5
225B...	....do...	1.0309	4.9	13.75	8.85	.70	....	.139	Trace.	42.0
226B...	Aug. 30	1.031	4.6	13.24	8.64	.72	....	.156	.07	41.0
227B...	....do...	1.0294	4.5	12.47	7.97	.66	....	.125	.07	39.0
228B...	....do...	1.0315	4.2	12.74	8.54	.70	....	.129	.33	40.1
229B...	....do...	1.033	4.2	12.03	7.83	.68	....	.149	Trace.	41.0
230B...	....do...	1.030	3.0	11.87	8.87	.67	....	.131	.13	40.0
231B...	....do...	1.0319	3.8	12.47	8.67	.69	....	.135	.07	40.1
232B...	....do...	1.0334	4.0	13.20	9.20	.72	....	.158	Trace.	42.0

[a] Traces of boric acid.
[b] Dirty bottle and pieces of leaves.
[c] Contained hair.
[d] Dirty bottle.
[e] Contained straw.
[f] Straw.

TABLE I.—*Analyses of milk sold in Washington and the District of Columbia*—Cont'd.

No. of sample.	Date.	Specific gravity.	Fat.	Total solids.	Solids not fat.	Ash.	Milk sugar.	Acidity.	Sediment.	Refractometer reading.
	1907.									
233B...	Aug. 30	1.030	5.0	13.28	8.28	.62	....	.132	Trace.	40.2
234B...	Sept. 3	1.0323	3.5	12.27	8.77	....	....	.198	.07	41.0
235B [a]..	....do...	1.0323	4.1	12.99	8.89	....	....	.169	Trace.	41.5
236B...	....do...	1.0317	4.5	13.32	8.82	....	....	.166	....	41.3
237B [a]..	....do...	1.0308	5.0	13.70	8.70	....	....	.148	.07	42.0
238B...	....do...	1.0327	4.4	13.45	9.05	....	....	.173	.07	43.0
239B...	....do...	1.0317	4.9	13.95	9.05	....	....	.171	Trace.	42.0
240B [a]..	....do...	1.0311	4.4	13.05	8.65	....	....	.157	.13	42.5
241B [a]..	....do...	1.0313	4.2	12.86	8.66	....	....	.162	.20	41.5
242B...	Sept. 4	1.0305	4.7	13.26	8.56	....	....	.162	.07	40.0
243B...	....do...	1.0309	4.1	12.64	8.54	....	....	.167	Trace.	39.0
244B...	....do...	1.032	4.0	13.05	9.05	....	....	.164	Trace.	40.0
245B...	....do...	1.0327	4.2	13.21	9.01	....	....	.155	....	40.1
246B...	....do...	1.033	5.2	14.39	9.19	....	....	.155	.13	41.0
247B...	....do...	1.0315	3.6	12.19	8.59	....	....	.189	.07	41.5
248B...	....do...	1.031	3.5	11.95	8.45	....	....	.198	.07	41.0
249B...	....do...	1.0327	3.8	12.74	8.94	....	....	.178	Trace.	42.0
251B...	Sept. 5	1.0281	3.8	11.60	7.80	.50	....	.135	Trace.	38.5
252B [b] .	....do...	1.0334	4.0	13.15	9.15	....	....	.173	.07	42.0
253B...	....do...	1.029	3.4	11.33	7.93	....	....	.135	.07	40.0
254B...	....do...	1.032	3.8	12.68	8.88	....	....	.162	Trace.	40.0
255B...	....do...	1.032	4.4	13.40	9.00	....	....	.228	Trace.	41.0
256B...	....do...	1.033	4.8	13.91	9.11	....	....	.144	.07	41.5
257B...	....do...	1.031	3.4	11.83	8.43	....	....	.140	Trace.	41.0
258B...	....do...	1.028	3.8	11.35	7.55	.59	....	.124	.13	38.0
259B...	Sept. 6	1.032	3.6	12.44	8.84	....	....	.162	Trace.	41.0
260B...	....do...	1.0325	4.3	13.41	9.11	....	....	.142	.60	41.0
261B...	....do...	1.0295	3.4	11.40	8.00	....	....	.142	.13	40.8
262B...	....do...	1.0314	4.6	13.37	8.77	....	....	.135	None.	41.1
263B...	....do...	1.0326	3.6	12.47	8.87	....	....	.144	.07	41.5
264B...	....do...	1.0296	3.9	12.08	8.18	....	....	.136	None.	40.0
265B...	....do...	1.0315	4.1	12.80	8.70	....	....	.128	None.	40.0
266B...	....do...	1.031	4.6	13.27	8.67	....	....	.140	.13	41.0
267B...	Sept. 9	1.0323	3.8	12.73	8.93	....	....	.153	.20	41.0
268B...	....do...	1.033	3.8	12.81	9.01	....	....	.178	.13	40.5
269B...	....do...	1.0335	3.9	13.05	9.15	....	....	.148	Trace.	41.0
270B...	....do...	1.0276	3.6	11.36	7.76	.654	....	.131	Trace.	39.0
271B...	....do...	1.032	3.6	12.32	8.72	....	....	.141	.07	39.0
272B...	....do...	1.0329	4.0	13.02	9.02	....	....	.149	Trace.	42.1
273B...	....do...	1.033	3.8	12.81	9.01	....	....	.160	Trace.	40.0
274B...	....do...	1.031	4.8	13.51	8.71	....	....	.149	.07	41.0
275B...	Sept. 10	1.0334	3.0	11.95	8.95	....	....	.164	None.	42.5
276B...	....do...	1.0324	4.0	12.90	8.90	....	....	.157	.07	42.0
277B...	....do...	1.0325	4.2	13.16	8.96	....	....	.409	Trace.	41.1
278B...	....do...	1.0316	3.5	12.10	8.60	....	....	.151	Trace.	41.0
279B...	....do...	1.0305	3.6	11.94	8.34	....	....	.139	None.	40.0
280B [c] .	....do...	1.031	4.4	12.55	8.15	....	....	.146	Trace.	41.0
281B...	....do...	1.0333	3.2	12.16	8.96	....	....	.149	.07	41.5
285B...	Sept. 11	1.0334	3.6	12.57	8.97	....	....	.164	None.	42.0
286B...	....do...	1.0325	4.2	13.19	8.99	....	....	.160	Trace.	41.0
287B...	....do...	1.0308	3.2	11.77	8.57	.88	....	.142	Trace.	40.0
288B...	....do...	1.0323	3.5	12.27	8.77	....	....	.180	None.	40.0

[a] Stale milk in bottle. [b] Dead fly. [c] Stale milk in bottle.

TABLE I.—*Analyses of milk sold in Washington and the District of Columbia*—Cont'd.

No. of sample.	Date.	Specific gravity.	Fat.	Total solids.	Solids not fat.	Ash.	Milk sugar.	Acidity.	Sediment.	Refractometer reading.
	1907.									
289B	Sept. 11	1.0325	4.4	13.40	9.00			.146	Trace.	41.0
290B	do	1.0271	3.0	10.92	7.92	.67		.133	.07	37.0
291B	do	1.0327	3.6	12.49	8.89			.148	.07	41.0
293B	do	1.0325	4.6	13.64	9.04			.162	Trace.	42.0
294B	Sept. 12	1.033	3.2	12.09	8.89			.144	Trace.	41.0
295B	do	1.0298	3.5	11.65	8.15			.122	.13	39.0
296B	do	1.0325	3.7	12.56	8.86			.139	.07	40.0
297B[a]	do	1.032	4.4	13.28	8.88			.144	.07	40.0
298B	do	1.0276	5.8	13.86	8.06			.142	None.	39.0
299B	do	1.0275	4.0	11.67	7.67			.131	.07	38.0
300B	do	1.032	3.8	12.56	8.76			.147	Trace.	40.0
301B	do	1.029	3.6	11.57	7.97			.136	Trace.	40.0
303B	Sept. 13	1.0325	4.4	13.40	9.00			.129	Trace.	41.5
304B	do	1.0321	4.0	13.05	9.05			.157	.13	42.0
305B	do	1.0311	4.2	12.81	8.61			.148	None.	41.0
306B	do	1.0317	3.7	12.35	8.65			.149	Trace.	40.0
307B	do	1.0328	4.2	13.24	9.04			.151	Trace.	41.5
308B	do	1.0279	4.8	12.73	7.93			.128	None.	39.0
309B	do	1.030	4.4	12.78	8.38			.151	None.	40.0
310B	do	1.030	3.8	12.06	8.26			.157	.07	40.0
311B	Sept. 16	1.026	8.9	17.18	8.28			.144	Trace.	39.0
312B	do	1.031	3.8	12.31	8.51			.139	Trace.	39.0
313B	Sept. 16	1.026	3.2	10.21	7.01	.608		.119	.07	37.0
314B	do	1.035	2.6	11.87	9.27			.115	.07	40.0
315B	do	1.0336	3.5	12.60	9.10			.167	.07	42.0
316B	do	1.030	4.2	12.54	8.34			.137	.07	39.0
317B	do	1.031	4.5	13.15	8.65			.135	.13	40.0
318B	do	1.0321	5.0	14.02	9.02			.160	None.	41.0
1C[a]	Sept. 18	1.031	4.6	13.27	8.67			.149	.07	42.0
2C[a]	do	1.0291	3.8	11.83	8.03			.122	.07	40.0
3C	do	1.0281	3.6	11.34	7.74			.122	Trace.	39.0
4C	do	1.0293	5.0	13.31	8.31			.113	.13	41.0
5C	do	1.0319	3.8	12.54	8.74			.175	Trace.	42.5
6C	do	1.0314	4.4	13.18	8.78			.135	.07	42.0
7C	do	1.0308	4.8	13.46	8.66			.131	Trace.	42.0
8C[a]	do	1.0333	3.7	12.76	9.06			.166	Trace.	42.0
9C	Sept. 19	1.027	3.8	11.48	7.68	.62		.115	.07	38.5
10C	do	1.0312	4.7	13.45	8.75			.133	.07	42.0
11C	do	1.0298	4.1	12.37	8.27			.140	.07	41.0
12C	do	1.0246	3.5	10.31	6.81	.514		.104	.07	36.0
13C	do	1.0296	3.5	11.60	8.10			.130	.07	39.0
14C	do	1.032	4.1	12.92	8.82			.149	Trace.	41.5
15C	do	1.0295	4.2	12.32	8.12			.139	.07	41.0
16C	do	1.0308	5.6	14.42	8.82			.126	.13	41.5
18C	Sept. 20	1.0269	4.8	12.66	7.86	.64		.137	.07	38.0
20C	do	1.0319	3.5	12.18	8.68			.146	.07	41.3
21C	do	1.0304	5.0	13.60	8.60			.181	.07	41.7
22C	do	1.0264	3.3	10.94	7.64	.60		.124	Trace.	37.5
23C	do	1.0275	4.6	12.40	7.80			.131	Trace.	39.5
24C	do	1.0327	3.8	12.74	8.94			.158	Trace.	41.7
25C	do	1.0307	4.4	12.96	8.56			.142	Trace.	41.0
26C	do	1.0241	3.6	10.44	6.84	.52		.099	Trace.	35.5

[a] Stale milk in bottle.

TABLE I.—*Analyses of milk sold in Washington and the District of Columbia*—Cont'd.

No. of sample.	Date.	Specific gravity.	Fat.	Total solids.	Solids not fat.	Ash.	Milk sugar.	Acidity.	Sediment.	Refractometer reading.
	1907.									
26½C	Sept. 23	1.031	3.4	11.84	8.44	......	......	.142	Trace.	40.5
27C	do	1.0314	3.6	12.18	8.58	......	......	.171	.07	41.0
28C	do	1.0323	4.0	12.88	8.88	......	......	.129	.07	41.5
29C	do	1.0298	5.2	13.69	8.49	......	......	.146	......	41.0
30C	do	1.0257	3.2	10.28	7.08	.49	......	.120	Trace.	36.0
31C	do	1.0315	4.8	14.18	9.38	.74	......	.165	.07	36.5
32C	do	1.0347	4.4	14.46	10.06	.86	......	.165	.07	44.5
33C	do	1.032	4.2	11.84	7.64	......	......	.151	Trace.	42.5
35C	Sept. 24	1.0315	4.2	12.92	8.72	......	......	.146	Trace.	41.5
36C	do	1.0319	3.8	12.54	8.74	......	......	.158		41.2
37C	do	1.0309	4.7	13.37	8.67	......	......	.128	.07	41.0
38C	do	1.0349	3.9	13.41	9.51	......	......	.212	None.	43.0
39C	do	1.0319	4.3	13.14	8.84	......	......	.151	.07	42.0
40C	do	1.0284	5.4	13.58	8.18	......	......	.166	None.	40.5
41C	do	1.0327	3.5	12.38	8.88	......	......	.157	None.	41.0
42C	do	1.032	3.6	12.32	8.72	......	......	.178	Trace.	41.0
44C[a]	Sept. 25	1.033	4.2	13.29	9.09	......	......	.155	.07	43.5
45C	do	1.0313	4.2	12.87	8.67	......	......	.151	Trace.	41.5
46C	do	1.0336	4.6	13.92	9.32	......	......	.157	None.	44.5
47C	do	1.0274	4.1	11.77	7.67	......	......	.124	None.	39.0
48C	do	1.0309	4.2	12.77	8.57	......	......	.158	Trace.	41.5
49C	do	1.0317	4.5	13.33	8.83	......	......	.149	None.	42.0
50C	do	1.0297	4.7	13.07	8.37	......	......	.128	.07	40.0
51C	do	1.033	4.8	14.01	9.21	......	......	.155	None.	43.0
52C	Sept. 26	1.0327	4.2	13.22	9.02	......	......	.160	Trace.	42.5
53C	do	1.0318	4.0	12.75	8.75	......	......	.158	Trace.	42.0
54C	do	1.0295	4.0	12.18	8.18	......	......	.140	None.	42.0
55C	do	1.0304	4.4	12.88	8.48	......	......	.137	.07	42.0
56C	do	1.0288	3.1	10.93	7.83	......	......	.124	Trace.	39.0
57C	do	1.0334	4.7	13.99	9.29	......	......	.153	None.	44.0
58C[a]	do	1.0303	4.9	13.46	8.56	......	......	.128	.07	42.0
59C	do	1.0303	3.8	12.15	8.35	......	......	.144	.07	41.0
60C[a]	Sept. 27	1.0329	5.0	14.23	9.23	......	......	.157	Trace.	43.0
61C	do	1.0334	4.5	13.76	9.26	......	......	.175	.07	43.0
62C	do	1.0314	4.6	13.38	8.78	......	......	.140	.13	41.5
63C	do	1.0334	4.2	13.39	9.19	......	......	.175	Trace.	42.5
64C	do	1.033	4.0	13.06	9.06	......	......	.160	Trace.	42.5
65C	do	1.032	4.4	13.28	8.88	......	......	.171	None.	42.5
66C	do	1.031	4.9	13.64	8.74		......	.131	.07	42.0
67C	do	1.0321	6.3	15.59	9.29		......	.167	Trace.	43.5

[a] Stale milk in bottle.

TABLE II. —*Showing Washington milks below standard, and those containing dirt, as previously reported to the health department.*

[The sample-numbers are those assigned by the D. C. Health Office. Each particular group of numbers represents all of the samples examined from any particular dairy.]

Sample numbers (D. C. health office) of milks examined.	Total number of milks examined.	Milks found below standard.	Total number of milks below standard.	Milks containing dirt.	Total number of milks containing dirt.
27 A, 80 A, 74 B, 159 B, 36 C, 14 B	6	74 B	1	80 A, 14 B, 159 B	3
50 A, 233 B, 264 B	3	50 A	1	50 A	1
72 A, 37 B, 200 B, 62 C, 317 B	5	37 B	1	37 B, 62 C, 317 B, 72 A.	4
57 B, 208 B, 315 B	3		0	57 B, 315 B	2
33 A, 95 B	2		0		0
29 A, 198 B	2		0	198 B	1
9 B, 32 B, 178 B, 246 B, 267 B, 60 C.	6		0	32 B, 172 B, 267 B, 246 B	4
42 C	1		0		0
41 A, 68 A, 25 B, 111 B, 161 B, 253 B, 265 B, 273 B, 301 B, 309 B.	10	41 A, 68 A, 253 B	3	25 B, 41 A, 111 B, 161 B, 253 B.	5
84 B.	1	84 B	1	84 B	1
38 A, 84 A, 127 B, 210 B, 15 C, 54 C.	6		0	127 B, 15 C	2
10 A, 101 B, 165 B, 259 B, 1 C	5		0	101 B, 165 B, 1 C	3
63 A	1		0		0
42 B.	1		0	42 B	1
49 B, 87 B	2		0	49 B, 87 B	2
53 A, 64 B, 27 C	3		0	64 B, 27 C	2
82 A, 90 B, 190 B, 294 B, 25 C	5	82 A, 294 B	2	82 A, 190 B	2
21 A, 81 A, 71 B, 119 B, 167 B, 217 B, 224 B, 231 B, 241 B, 35 C, 242 B.	11	119 B	1	119 B, 167 B, 231 B, 241 B, 242 B, 71 B.	6
7 A, 16 B, 12 B, 73 B, 61 B, 155 B, 277 B, 65 B, 39 C.	9	65 B, 61 B	2	39 C, 12 B, 16 B, 73 B, 155 B.	5
91 B, 260 B	2		0	91 B, 260 B	2
2 A, 46 C	2		0		0
28 A, 38 B	2		0	38 B	1
42 A, 299 B	2	299 B	1	299 B	1
11 A, 5 B, 85 B, 163 B, 41 C	5		0	85 B, 163 B	2
46 A, 2 B, 86 B, 185 B, 51 C	5		0	86 B, 185 B	2
77 A.	1		0	77 A	1
1 A, 58 A, 22 B, 124 B, 228 B, 303 B, 4 C.	7		0	124 B, 228 B, 4 C	3
52 B	1		0	52 B	1
75 A, 46 B, 121 B	3		0	46 B, 75 A	2
51 B, 184 B, 196 B, 293 B, 64 C	5		0	184 B	1
107 B	1		0		0
24 B.	1	24 B	1	24 B	1
5 A, 64 A, 20 B, 93 B, 225 B, 312 B	6		0	64 A	1
16 A, 81 B, 137 B, 212 B, 22 C	5	16 A, 22 C	2	81 B, 137 B	2
13 A, 83 A, 89 B, 149 B, 263 B, 24 C	6		0	263 B	1
12 A, 67 A, 180 B	3		0		0
76 A, 50 B, 183 B, 65 C	4		0	50 B, 183 B, 76 A	3
63 B, 164 B, 305 B, 29 C	4		0	63 B	1
43 B	1		0		0
59 C	1		0	59 C	1
182 B	1		0	182 B	1
113 B, 306 B, 16 C, 17 B	4		0	113 B, 16 C, 17 B	3
25 A, 307 B	2		0		0
146 B, 295 B	2		0	146 B, 295 B	2

TABLE II.—*Showing Washington milks below standard, and those containing dirt, as previously reported to the health department.*—Continued.

Sample numbers (D. C. health office) of milks examined	Total number of milks examined.	Milks found below standard.	Total number of milks below standard.	Milks containing dirt.	Total number of milks containing dirt.
138 B, 56 C	2	138 B, 56 C	2		0
23 A, 108 B, 216 B, 223 B, 232 B, 240 B, 247 B, 252 B, 262 B, 272 B, 278 B, 288 B, 300 B, 304 B, 11 C, 37 C.	16	108 B	1	240 B, 247 B, 252 B, 108 B, 216 B, 11 C, 223 B, 304 B, 37 C.	9
39 A, 78 A, 85 A, 6 B, 76 B, 94 B, 132 B, 144 B, 256 B, 297 B.	10	78 A	1	78 A, 85 A, 76 B, 94 B, 132 B, 144 B, 56 B, 297 B.	8
37 A, 29 B, 110 B, 187 B, 53 C	5		0	187 B, 37 A	2
44 A, 80 B, 136 B, 213 B, 21 C	5		0	80 B, 136 B, 21 C, 44 A.	4
130 B, 261 B, 298 B	3	261 B	1	130 B, 261 B	2
57 A, 66 B, 104 B, 172 B, 230 B, 9 C, 47 C.	7	57 A, 230 B, 9 C	3	104 B, 172 B, 230 B, 9 C, 57 A.	5
251 B, 18 C	2	251 B	1	18 C	1
98 B, 188 B, 13 C	3		0	188 B, 13 C	2
78 B, 145 B, 238 B	3		0	238 B	1
62 B, 67 C	2		0		0
35 A, 27 B, 152 B, 214 B	4		0	35 A, 152 B, 214 B	3
54 A, 6 C, 162 B	3		0	6 C, 54 A, 162 B	3
106 B, 229 B	2	106 B	1	106 B	1
19 A, 48 A, 15 B, 115 B, 271 B, 26½ C.	6	48 A, 26½ C	2	115 B, 271 B, 19 A, 48 A.	4
23 B, 117 B, 32 C	3		0	23 B, 117 B, 32 C	3
56 B, 83 B, 158 B, 206 B, 270 B.	5	56 B	1	83 B, 158 B, 206 B	3
17 A, 79 A, 13 B, 35 B, 92 B	5	79 A	1	79 A, 13 B, 35 B, 92 B.	4
20 A, 54 B, 142 B, 218 B, 287 B, 311 B.	6	287 B	1	54 B, 218 B	2
102 B, 275 B	2	275 B	1		0
36 B, 204 B	2		0	36 B, 204 B	2
280 B, 44 C	2		0	44 C	1
139 B, 291 B	2		0	139 B, 291 B	2
128 B, 55 C	2		0	128 B, 55 C	2
105 B, 168 B, 7 C, 49 C	4		0		0
24 A, 26 B, 192 B, 313 B, 26 C, 30 C.	6	24 A, 30 C, 313 B, 26 C.	4	26 B, 192 B, 313 B, 24 A.	4
44 B, 220 B	2		0	44 B, 220 B	2
40 A, 40 B, 140 B, 243 B, 61 C	5		0	40 B, 61 C, 40 A	3
9 A, 52 A, 4 B, 100 B, 166 B, 202 B, 237 B.	7	52 A	1	4 B, 100 B, 166 B, 202 B, 237 B.	5
191 B, 314 B	2	314 B	1	191 B, 314 B	2
8 A, 11 B	2		0		0
103 B, 276 B	2		0	103 B, 276 B	2
43 A, 79 B, 221 B, 58 C	4		0	79 B, 58 C, 43 A	3
31 C	1		0	31 C	1
47 A, 99 B, 179 B, 8 C, 38 C	5	99 B	1	99 B, 47 A	2
55 B.	1		0	55 B	1
26 A, 61 A, 18 B, 34 B, 59 B, 116 B, 193 B, 45 C.	8		0	34 B, 59 B	2
32 A, 97 B, 154 B, 269 B	4		0	154 B, 32 A	2
75 B, 157 B	2		0		0

TABLE II.—*Showing Washington milks below standard, and those containing dirt, as previously reported to the health department*—Continued.

Sample numbers (D. C. health office) of milks examined	Total number of milks examined	Milks found below standard	Total number of milks below standard.	Milks containing dirt.	Total number of milks containing dirt.
18 A, 33 B, 150 B, 268 B	4	18 A, 33 B	2	268 B, 150 B, 33 B, 18 A.	4
59 A, 114 B, 169 B, 227 B, 308 B, 2 C.	6		0	114 B, 169 B, 227 B, 2 C, 59 A.	5
49 A, 148 B	2		0		0
147 B, 289 B	2		0	147 B	1
133 B, 281 B	2	281 B	1	281 B	1
134 B, 234 B	2		0	134 B, 234 B	2
36 A, 28 B, 112 B, 186 B, 219 B, 285 B, 14 C.	7	219 B	1	28 B, 112 B, 219 B	3
235 B	1		0		0
69 A	1		0	69 A	1
41 B	1		0		0
60 A, 70 B, 205 B, 245 B, 40 C	5	70 B	1	70 B, 205 B	2
15 A, 129 B, 211 B	3		0	211 B	1
30 A, 53 B, 197 B, 63 C	4		0	197 B, 30 A	2
58 B, 156 B, 209 B	3		0	156 B, 209 B	2
27 A, 51 A, 39 B, 153 B, 199 B, 52 C.	6		0	153 B, 51 A	2
7 B, 131 B, 215 B, 222 B, 239 B, 248 B, 257 B, 274 B, 290 B, 296 B, 310 B.	11	222 B, 257 B, 290 B.	3	7 B, 131 B, 215 B, 222 B, 248 B, 274 B, 290 B, 296 B, 310 B.	9
181 B	1		0	181 B	1
3 A, 74 A	2		0		0
14 A, 30 B, 82 B, 135 B, 189 B, 12 C, 23 C.	7	30 B, 12 C.	2	82 B, 135 B, 189 B, 12 C.	4
3 B, 174 B, 254 B, 279 B, 3 C	5	3 B	1	3 B, 174 B	2
34 A, 96 B, 141 B	3		0	96 B	1
55 A, 69 B	2	55 A	1	55 A	1
1 B.	1		0		0
70 A, 72 B, 88 B, 120 B, 194 B, 258 B, 316 B, 66 C, 15 A.	9	258 B	1	88 B, 194 B, 258 B, 316 B, 66 C, 70 A.	6
31 A, 71 A, 8 B, 122 B, 195 B, 249 B, 28 C.	7	31 A	1	195 B, 28 C, 71 A	3
236 B	1		0		0
66 A, 77 B, 176 B, 266 B, 20 C	5		0	266 B, 20 C, 66 A	3
68 B, 123 B, 173 B, 5 C	4	173 B	1	123 B, 173 B	2
62 A, 40 B, 60 B, 126 B, 50 C	5		0	50 C	1
6 A 65 A, 19 B, 109 B, 160 B, 33 C.	6	6 A	1	19 B, 109 B, 160 B, 65 A	4
4 A, 21 B, 203 B	3		0	21 B, 203 B	2
56 A, 67 B, 171 B, 207 B, 226 B, 244 B, 286 B, 48 C	8		0	67 B, 171 B, 207 B, 226 B, 56 A.	5
57 C	1		0		0
45 A, 73 A, 175 B, 201 B, 255 B, 318 B, 10 C.	7		0	201 B, 10 C, 73 A	3
Totals	452		55		242

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11. THE NUMBER OF BACTERIA IN MILK AND THE VALUE OF BACTERIAL COUNTS.

THE NUMBER OF BACTERIA IN MILK AND THE VALUE OF BACTERIAL COUNTS.

By Milton J. Rosenau,

Director, Hygienic Laboratory, Public Health and Marine-Hospital Service.

Milk delivered in cities contains a vast number of bacteria. For instance, the general milk supply of Washington averaged 11,270,000 per cubic centimeter in the summer of 1907; and 22,134,000 during the summer of 1906. The milk of many other cities also is excessively rich in bacteria.

Such enormous numbers mean but little to our minds. If we make comparisons we find that few substances contain such myriads of germ life as is often found in milk. Compared with sewage, for instance, a fluid which is popularly and rightly supposed to teem with germ life, it will almost always be observed that milk when it is consumed is richer in bacteria by far than the sewage of our large cities.[a]

Sewage of-	Average for	Bacteria per cubic centimeter
Boston, Mass.[a]	1894 to 1901	2,800,000
London, England[b]	1894 to 1901	2,000,000 to 11,000,000
London, England[c] (crude sewage)	1898	3,500,000 to 4,000,000
Lawrence, Mass.[d]	Sept. 24 to Oct. 24, 1890	3,034,000
St. Mary's, Ohio[e]	16 samples, 1907	5,600,000
Westerville, Ohio[e]	16 samples, 1907	2,350,000
Marion, Ohio[e]	16 samples, 1907	239,000

[a] Winslow and Belcher. Changes in the bacterial flora of sewage during storage.

[b] Laws and Andrews. Report on the result of investigations of the micro-organisms of sewage. Rep. London Co. Council, Dec. 13, 1894.

[c] Clowes, F. Report on the bacteriological examination of London crude sewage. First Rep. London Co. Council, June 16, 1898.

[d] State Board Health Mass., Rep. 1890, p. 35.

[e] Kellerman, Pratt and Kimberly. The disinfection of sewage effluents for the protection of public water supplies. U. S. Bur. Plant Industry, Bull. 115, 1907.

So far as numbers are concerned, they need not greatly alarm us, for we know that disease is due to agencies and conditions other than merely the presence of enormous numbers of bacteria. By universal consent, however, milk containing excessive numbers of bacteria is unsuitable for infant feeding. The tender mucous membrane of infants is very susceptible to bacteria and their products, and a large proportion of the summer complaints of infants has been traced to the use of bacteria-laden milk. As we grow older it seems that the gastro-intestinal mucous membrane becomes comparatively immune, or resistant to bacterial action.

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[a] Russell, H. L. Outlines of Dairy Bacteriology, 1896.

If milk were a transparent fluid the enormous growth of bacteria found in market milk would be plainly visible to the naked eye. A similar amount of bacterial growth in broth, gelatine, beer, jelly, or other clear substance, would render such food unsightly, and it would be generally regarded as unfit for use on account of the evidence of fermentative and putrefactive changes.

The number of bacteria in milk is not so important from a public health standpoint as the kind and nature of the bacterial products. But with cleanliness and the liberal use of ice the total number of bacteria can be kept down, and this affords a mode of protection against the dangerous species and their toxic products. Milk containing few bacteria will contain proportionately few or no harmful varieties. Most of the specific pathogenic bacteria which sometimes contaminate milk, grow best at the body temperature and not at all at the low temperatures at which milk must be kept in order to keep the total bacterial count down.

Park[a] raises the question—

> Are even these enormous numbers of bacteria in milk during hot weather actually harmful? Here we have only to refer to universal clinical experience, that a great number of children in cities sicken on the milk supplied in summer, that those put on milk which is sterile or contains few bacteria as a rule mend rapidly, while those kept on the impure milk continue ill or die.
>
> Our knowledge is probably as yet insufficient to state just how many bacteria must accumulate to make them noticeably dangerous in milk. Some varieties are undoubtedly more harmful than others and we have no way of restricting the kinds that will fall into milk except by enforcing cleanliness. We have also to consider that milk is not entirely used for some twelve hours after being purchased, and that during all this time bacteria are rapidly multiplying, especially where, as among the poor, no provision for cooling it is made. Slight changes in the milk which to one child would be harmless would in another produce disturbances which might lead to serious disease. A safe conclusion is that no more bacterial contamination should be allowed than it is practical to avoid. Any intelligent farmer can use sufficient cleanliness and apply sufficient cold, with almost no increase in expense, to supply milk twenty-four to thirty-six hours old which will not contain in each cubic centimeter over 50,000 to 100,000 bacteria, and no milk containing more bacteria should be sold.

Judged by the colonies that develop upon agar plates, the number of bacteria in milk increases every time it is handled. Separator milk contains more than the original milk. The same is true of filtered milk. Milk strained through gauze or cotton, or filtered through gravel or any other device, while it looks clean, always contains more bacteria than before it has been "purified." This is due to the fact that, while the visible particles of dirt are held back, the particles of manure, dirt, and bacterial clusters are broken up. Further, unless the most painstaking technical precautions are taken, milk receives fresh bacterial contamination every time it is poured from one vessel to another or is handled in any other way.

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[a] Park, W. H.: The great bacterial contamination of the milk of cities, can it be lessened by the action of health authorities? Journ. Hyg., vol. 1, 1901, p. 391.

THE INITIAL CONTAMINATION OF MILK.

Now that we know that milk freshly drawn from the udder under ordinary circumstances always contains bacteria, it is of practical importance to determine their number and kind.

Sedgwick and Batchelder,[a] 1892, found that with moderate precautions on the part of the milker the number of bacteria in fresh milk may not exceed 500 to 1,000 per cubic centimeter, but when the ordinary flaring milk pail is used, with more or less disturbance of the bedding and shaking of the udder, as many as 30,000 bacteria have been counted in one cubic centimeter.

MacConkey,[b] however, finds that with ordinary care and cleanliness it is possible to obtain milk which when freshly drawn contains less than 1,500 organisms per cubic centimeter; and, further, that such milk should not contain gas-forming organisms in less than 50 cubic centimeters.

Comparing these results with the work of others, we find that Park,[c] 1901, found the average bacterial content of the milk from six separate cows examined five hours after collection to be 6,000 per cubic centimeter, the lowest count being 400, and of 25 cows of which the milk was tested immediately after drawn it was 4,550.

Burr,[d] 1902, also taking every reasonable precaution, found 500 organisms per cubic centimeter in the milk of a single cow.

Von Freudenreich,[e] 1902, thought it would be easy to carry out strict asepsis and thus obtain a bacteria-free milk; but he soon came to the conclusion that this was impossible. He found that milk always contained 250 to 300 organisms per cubic centimeter, even though the milker's hands and the teats were washed first with soft soap and sterile water and then with servatol soap and sterile water, and finally with sterile water alone and dried on a sterile towel. The milker's hands were smeared with lanoline and the first milk rejected. The bacterial content of the mixed milk of 28 cows milked in this way varied from 65 to 680 organisms per cubic centimeter.

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[a] Sedgwick, William T., and Batchelder, John L.: A bacteriological examination of the Boston milk supply. Boston Med. and Surg. Journ., vol. 126, 1892, p. 25–28.

[b] MacConkey: A contribution to the bacteriology of milk. Journ. of Hyg. vol. 6, 1906, p. 385.

[c] Park, William H.: The great bacterial contamination of the milk of cities, can it be lessened by the action of the health authorities? Journ. Hyg., vol. 1, 1901. p. 391.

[d] Burr, Rollin H.: The source of the acid organisms of milk and cream. Cent. f. Bakt., 2 Abt., vol. 8, 1902, p. 236.

[e] Von Freudenreich, Ed.: Milchsäurefermente und Käsereifung. Cent. f. Bakt., 2 Abt., vol. 8, 1902, p. 674.

Von Freudenreich and Thöni,[a] 1903, from a further series of similar experiments conclude that freshly drawn milk, even when the most careful precautions are taken against contamination, always contains bacteria; that these are mostly cocci and that they come from the udder.

Continuing his experiments, Von Freudenreich,[b] 1903, states that he examined the udders and the milk in the udders of 15 cows, in 13 cases immediately after slaughtering. The organisms were mostly cocci. *B. lactis acidi* was only met with once. In 3 cases the ducts were diseased and in these cases the diseased tissues contained fewer organisms than usual. *B. coli* was never found. He mentions that Boekhout and De Vries drew milk directly from the udder with a sterile canula and always got a growth from it.

Lux,[c] 1904, examined milk drawn without aseptic precautions. Two hundred and sixty cow-milk and 95 goat-milk samples were analyzed. The average number of bacteria per cubic centimeter was 1,395, which were mostly nonpathogenic cocci.

Henderson,[d] 1904, examined seven normal udders and obtained growth in 76 per cent of the cultures made, the organisms being staphylococci, streptococci, and pseudo-diphtheria bacilli. No organisms found were pathogenic to laboratory animals.

Willem and Miele,[e] 1905, obtained a milk containing 2.5 bacteria per cubic centimeter. The milking was done in a special place, which was kept as aseptic as possible. The greatest care was taken to insure the cows being clean. The udder and teats were washed before each milking with soap and boiled water or an aseptic solution.

From the examples quoted we see that it is practically impossible to obtain bacteria-free milk, but that the organisms in carefully collected milk are not pathogenic to the usual laboratory animals. We may allow, then, that the presence of such organisms in reasonable number would not render a milk harmful to man. Lux's experiments have shown that with very ordinary care it is possible to obtain a milk containing on an average 1,400 bacteria per cubic centimeter, and it is obvious that with some trouble the number may be reduced.

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[a] Von Freudenreich, Ed., and Thöni, J.: Ueber die in der normalen Milch vorkommenden Bakterien und ihre Beziehungen zue dem Kasereifungsprozesse. Cent. f. Bakt., 2 Abt., vol. 10, 1903, p. 305.

[b] Von Freudenreich, Ed.: Ueber das Vorkommen von Bakterien im Kuheuter. Cent. f. Bakt., 2 Abt., vol. 10, 1903, p. 401.

[c] Lux, Arthur. Ueber den Gehalt der frisch gemolkenen Milch an Bakterien. Cent. f. Bakt., 2 Abt., vol. 11, 1903, p. 195.

[d] Henderson, J.: Journ. roy. san. inst., vol. 25, 1904, p. 563.

[e] Willem and Miele. Procédé pour l'obtention du lait au aseptique. Compt. Rend. du 13 Cong. internat. d'hyg., Brux., 1903, vol. 3, p. 67.

The work of Park,[a] 1901, Nicolle and Petit,[b] 1903, Conn and Esten,[c] 1904, Koning,[d] 1905, Harrison,[e] 1905, and others has shown that if milk be rapidly cooled to 11° C. (50° F.) or below, very little, if any, multiplication of micro-organisms takes place for some twelve hours. Therefore Park's suggested average standard of not more than 12,000 bacteria per cubic centimeter in warm and 5,000 in cold weather for freshly drawn milk seems a generous standard and one which, with a little care, should be easily attained.

It is necessary to note that "separator milk" must not be judged by the same standard as fresh milk, for Severin and Budinoff,[f] 1905, and Severin,[g] 1905, have shown that even when every possible precaution is taken against contamination, the milk issuing from the separator always contains many more bacteria than it did before it passed into the separating chamber. Severin suggests that the mechanical movement completes the separation of bacteria which were only partially divided when they entered the machine.

Moore[h] concludes from a large mass of data that freshly drawn fore milk contains a variable but generally enormous number of bacteria, but only a few species, the last milk containing as compared with the fore milk very few micro-organisms.

Russell[i] found that the mixed milk of a herd that is kept with any reasonable degree of cleanliness, if examined immediately after milking, usually will not contain more than 5,000 to 20,000 germs per cubic centimeter.

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[a] Park, Wm. H.: The great bacterial contamination of the milk of cities. Can it be lessened by the action of health authorities? Journ. Hyg., vol. 1, 1901, p. 391.

[b] Nicolle, C., and Petit, P.: Etude expérimentale sur la question du lait à Rouen. Rev. med. de Normandia, 1903. Rev. Bull. de l'Inst. Pasteur, vol. 2, 1904, p. 552.

[c] Conn, H. W., and Esten, W. M.: The effect of different temperatures in determining the species of bacteria which grow in milk. Storrs Agric. Exper. Sta., 15th ann. rep., June 30, 1904, pp. 27–88.

[d] Koning: Biologische und biochemische Studien über Milch. Milchwirtschaftl Centblt., vol. 1, 1905; Rev., Cent. f. Bakt., 2 Abt., vol. 14, 1905, p. 424.

[e] Harrison, F. C.: A comparative study of sixty-six varieties of gas-producing bacteria found in milk. Cent. f. Bakt., 2 Abt., vol. 14, 1905, p. 359.

[f] Severin, S., and Budinoff, L.: Ein Beitrag zur Bakteriologie der Milch. Cent. f. Bakt., 2 Abt., vol. 14, 1905, p. 463.

[g] Severin, S.: Vermindert die Zentrifugierung die Bakterienzahl in der Milch? Cent. f. Bakt., 2 Abt., vol. 14, 1905, p. 605.

[h] Moore: U. S. Bur. Animal Indus., 1895–6.

[i] Russell, H. L.: Outlines of dairy bacteriology, 1896, p. 59

I have found the milk obtained by careful methods from separate cows to contain the following number of bacteria per cubic centimeter immediately after milking: 60, 160, 400, 400, 500, 500, 8,300.

All these counts are evidently too low, for the reason that not all the bacteria produce visible colonies upon agar plates, and further each colony does not necessarily represent the growth from one microorganism. Rosenau and McCoy have shown elsewhere (upon the germicidal property of milk this Bulletin, p. 447) that the bacteria in milk are apt to agglutinate into clusters.

LEGAL STANDARDS.

The first attempt to make a standard for the bacteriological content of milk was undertaken by the New York board of health, which, in 1900, believed it was not necessary for any milk sold in New York to contain over 1,000,000 bacteria per cubic centimeter. It was found, however, practically impossible to enforce such a standard for the city of New York on account of the complexity and enormous volume of the milk trade of that city. The principal difficulty was to place the responsibility when milk was found to contain an excessive number of bacteria, as the milk passed through so many hands before it was delivered to the consumer.

Boston, on the other hand, made a strict standard of 500,000 bacteria per cubic centimeter, which was legalized by the board of health June 6, 1905, in article 6, section 1, of the Regulations for the Sale and Care of Milk. According to Jordan,[a] the adoption of a bacteriological standard by the Boston board of health has been decried and the subject of scoffing, but the example of that city has since been followed by other municipalities, until now nearly 20 cities are conducting bacteriological investigations of milk supplies. This outcome is fortunate, for from multiplication of work of this character great progress may be expected.

Goler,[b] health officer of the city of Rochester, issued a circular to all milk producers supplying that city, informing them that thereafter 100,000 bacteria per cubic centimeter would be made a maximum standard.

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[a] Jordan, James O.: Boston's campaign for clean milk. Journ. Am. Med. Assn., vol. 49, Sept. 28, 1907.

[b] Goler, George W.: Municipal regulation of the milk supply. Trans. Soc. on Hyg. & San. Science, A. M. A., June 1907, p. 251.

Bitter[a] believes that no milk should be sold in cities containing more than 50,000 bacteria per cubic centimeter.

Park[b] states that any intelligent farmer can use sufficient cleanliness and apply sufficient cold with almost no increase in expense to supply milk twenty-four to thirty-six hours old which will not contain in the maximum over 50,000 to 100,000 bacteria per cubic centimeter, and that no milk containing more bacteria than this should be used.

The above figures apply to standards that have been set on market milk. So far as milk for infant feeding and other clinical purposes is concerned, the standard established by Coit of 10,000 bacteria per cubic centimeter as a maximum seems, by almost unanimous consent, to be the best. Some communities have adopted a second grade of milk known as "inspected" milk from tuberculin-tested cattle and obtained under cleanly conditions, and not containing over 100,000 bacteria per cubic centimeter.

The number of bacteria, therefore, allowable in milk depends upon the purposes for which it is used and varies somewhat with the locality. It is evidently easier to obtain milk containing fewer bacteria in small communities with a near-by supply and in cold climates, than it is in larger cities with inevitable delays in transportation or in southern latitudes.

As a general rule it may be stated that "certified" milk should never exceed 10,000 bacteria per cubic centimeter, "inspected" milk not over 100,000, and health officers should aim to keep the general milk supply below the 100,000 mark.

THE PRACTICAL VALUE OF BACTERIAL EXAMINATIONS OF MILK.

The activities of our health officers were at first directed almost exclusively to the prevention of sophistication of milk, detected by chemical methods, to the neglect of the valuable information obtained from bacterial examinations.

The addition of water to milk and the extraction of cream are fraudulent practices, but, as a rule, have only a secondary bearing upon the public health. The bacteriologic examination of milk gives us a clew to the cleanness of the methods employed, the temperature, and the age of the milk. The health officer who has the advantage of bacteriologic assistance knows that the milk of dairies containing excessive numbers of bacteria is dirty, old, or warm.

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[a] Bitter, H.: Versuche über das Pasteurisiren der Milch. Zeit. f. Hyg., vol. 8, 1890, p. 240.

[b] Park, William H., and Bebb, Rose A. The great bacterial contamination of the milk of cities. Can it be lessened by the action of health authorities? N. Y. Univ. Bull. Med. Sci., vol. 1, 1901.

With a bacteriologic count as a guide it is comparatively easy to determine the cause of the trouble and to institute proper means to correct it. The enumeration of bacteria in milk is, therefore, one of the readiest and cheapest methods at the disposal of health officers to determine the general sanitary quality of the market milk supply. The laboratory results serve not only as a guide to direct the efforts of the health officer, but confirm the conclusions arrived at from an inspection of the dairies and dairy farms.

While the bacteriological examination of milk has its uses, it also has distinct limitations. From a practical standpoint the long time required to obtain results is its greatest drawback. The qualitative determinations of the bacterial species in milk is too complex and difficult a method to adopt as a routine procedure. It is otherwise with quantitative counts. These determinations are comparatively easy and are of invaluable assistance to the progressive dairyman in controlling his methods and in discovering just which cow, what person, or what part of the industry is at fault when things go wrong.

It is comparatively easy to make bacterial counts of milk, and for practical purposes the method may soon be learned even by one not skilled in bacteriologic technique. Dairymen will find it to their advantage to make agar plates and roughly estimate the number of bacteria, not only of their finished product, but from individual cows and during various stages in the handling of the milk.

In fact, a number of progressive dairymen are already using bacteriologic counts of their milk in order to improve the supply. In Boston, Jordan tells us that in 1906, six milk firms made over 27,000 such examinations.

In Rochester, Goler[a] has obtained a reduction in the average bacterial count of the milk supply of that city from 837,000 per cubic centimeter in 1900 to 200,000 in 1903. In 1900, 26 per cent of the samples examined contained over 5,000,000 bacteria per cubic centimeter; in 1903 only 4 per cent contained over 5,000,000. At the time the city milk supply contained an average of 235,000 bacteria per cubic centimeter, the milk that was procured under a process of certification and education contained but 14,000 bacteria per cubic centimeter for the same period.

In Washington the bacteriological examinations made in the Hygienic Laboratory and submitted to the dairies by the local health officer have stimulated the dairymen to use more ice, with the result that during the summer of 1907 the average temperature of 316 samples of milk examined was 2.3° C. lower than during the corresponding term for 1906, and the average number of bacteria per cubic centimeter was cut in half. Convinced of the practical advantages of the bacteriological control of milk, one progressive dairyman in Washington has employed a competent bacteriologist to assist him in marketing a better quality of milk.

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[a] Goler, G. W.: The influence of the municipal milk supply upon the deaths of young children. N. Y. State Journ. Med., vol. 3, 1903, p. 493.

One great advantage accruing from the bacteriological control of milk is that it affords an opportunity to exclude the milk of diseased cows. Cows frequently suffer with diseases of the udder; in fact, garget or mammitis is the most common of all bovine diseases. Milk from inflamed udders containing pus-producing organisms (streptococci) is believed by some to be more important than the peptonizing species, about which much has been said since the work of Flügge.

Fresh milk from cows with diseased udders contains an excessive number of streptococci and pus cells or an excess of pus cells alone. So far as we know, such milk is dangerous for infant feeding. While not all agree with this view, nor is there any agreement concerning what constitutes an excessive number of streptococci and pus cells in milk, the facts have been put to practical use by Jordan in Boston. There, milk "infected" with excessive numbers of streptococci or an excess of pus was traced back to the cow, with the result that thirty-one diseased cows supplying milk to Boston in 1906 were found and eliminated. Most of the animals had mammitis or garget; some had ulcerated teats, some had recently calved, and others were approaching the calving period, etc.[a]

BACTERIAL COUNTS OF WASHINGTON MILK.

METHODS.

The number of bacteria found in any given sample of milk will vary with the methods used. It is not possible by any known method in bacteriology to determine the exact number of live bacteria in a sample of milk. The counts obtained are always below the actual number present. This is due to a number of reasons. First of all the bacteria stick together in groups and clusters; some are held together by adhesive membranes in pairs, chains, or masses. It is therefore evident that a single colony on a plate may not represent the growth from a single micro-organism.

It is impossible to obtain a medium, temperature, and other conditions suitable to the requirements of all bacteria. Some grow best at high temperatures, others at low; some prefer acid, others alkaline media; some need oxygen, which is fatal to others, etc.

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[a] Thirty-fifth ann. rep. city of Boston, Health Dept., 1906.

After careful consideration of the subject the following methods have given satisfactory results in this laboratory:[a]

The samples were always collected in the original containers, either pint or quart bottles being purchased for our purposes. Some of these samples were obtained from the wagon on the street, others from the dairy, and still others were obtained from houses in various parts of the city, at once after being delivered in the usual course of trade. It is therefore believed that the samples examined fairly represent the average milk obtained by the householder. The samples were collected early in the morning and at once placed in a metal container filled with cracked ice. From six to eight samples were collected each morning from various parts of the city, and rarely more than two hours elapsed from the collection of the first sample to the time it was received in the laboratory. The temperature was taken with a good thermometer at the time the sample was collected, but always from a different bottle, which was afterwards used for chemical purposes.

It was noted that after the milk stood on ice for some time that there might be a difference of 6 to 8 degrees between the top and the bottom layers of the milk in a pint bottle. The milk was always shaken well in order to mix the cream and to help break up the bacterial clumps before the bottle was opened, which was done with the usual bacteriologic precautions. For ordinary market milk the following dilutions were made:

1 cubic centimeter milk + 99 cubic centimeters sterile water.

0.1 cubic centimeter of this was used for the first plate, which represented 1:1,000.

0.5 cubic centimeter of the first dilution was then added to 49.5 cubic centimeters of sterile water. One cubic centimeter of this dilution when plated represented 1:10,000, and 0.1 cubic centimeter of this dilution represented 1:100,000.

The dilutions were vigorously shaken at least twenty-five times in accordance with the standard methods for water analysis in order to obtain uniform suspension of the bacteria. Sterile distilled water was used as a diluent.

The final dilution was measured directly into a petri dish and agar poured at a temperature of between 40° and 45° C. in the usual way.

After the plates were well set, they were grown at 37° C., which temperature appears not only to favor the maximum growth of bacteria ordinarily found in the milk, but has the additional advantage of favoring the kinds of bacteria belonging to the pathogenic class. The plates were counted at the end of twenty-four hours, although by that time the maximum growth had not appeared. Only those colonies were counted which were visible to the naked eye or could be seen with a low-power magnifying glass. Three plates were always made from each sample, one from each dilution. Plates that became spoiled owing to spreading of the surface growths over them, irregular distribution, or excessive numbers, were discarded. The counts were always taken when possible from plates containing 200 or less bacteria per plate, the reading being reduced to round numbers.

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[a] Since writing this article the committee on standard methods of bacterial milk analysis have presented a preliminary report, which appeared in the American Journal of Public Hygiene, vol. 17, November, 1907, pp. 331-364. At the Winnipeg meeting of the American Public Health Association in September, 1908, the committee presented a report of further progress, an abstract of which appeared in the American Journal of Public Hygiene for November, 1908, p. 425. (See also Heineman and Glenn's recent article on "A comparison of practical methods for determining the bacteriological content of milk," Journ. Infectious Diseases, vol. 5, Oct. 20, 1908, pp. 412-420.)

The composition of the media used for this work was 1.5 per cent agar and an acidity of + 1.5 to phenolphthalein as an indicator.

In accordance with this method a number of samples of milk bought on the open market in Washington were examined in the Hygienic Laboratory, P. H. & M. H. S., during the summer months of 1906-7, the results of which appear in the following tables:

Results of bacterial counts of market milk in Washington, 1906 and 1907.

Name.	Date.	Samples obtained at	Temperature of milk when obtained (° C.).	Number of bacteria per cubic centimeter.
Ag.	July 10, 1907		9	8,000
	July 22, 1907	Wagon..	10.5	69,000
	Aug. 5, 1907	Dairy...	9	2,680,000
	Aug. 19, 1907	..do..	6	170,000
All	July 15, 1907		17	2,240,000
	July 30, 1907	Providence Hospital.	22	5,150,000
	July 31, 1907	..do..	11	111,000,000
	Aug. 30, 1907	Dairy...	23	190,000
Al...	July 11, 1907		18.2	1,700,000
	Aug. 26, 1907	Dairy...	7.5	1,090,000
Alt..	Aug. 1, 1907	..do....	19.5	22,500,000
	Aug. 27, 1907	..do....	14	2,400,000
Arn.....	July 12, 1907		26.5	26,000,000
	Aug. 7, 1907	Dairy..	24	870,000
Art...	July 24, 1907	..do..	20	350,000
	July 29, 1907	Wagon..	20	2,560,000
	Aug. 20, 1907	.....do..	13	950,000
Av.....	Aug. 30, 1906	Dairy...	14.5	18,300,000
B.	July 13, 1907		16	3,660,000
	Aug. 6, 1907	Wagon	27	1,000,000
Ba	July 16, 1907	..do	21	2,800,000
	Aug. 2, 1907	..do	19	880,000
Bee..	July 12, 1907		10	700,000
	July 22, 1907		12	2,180,000
	Aug. 14, 1907	Dairy	7	11,700,000
	Aug. 28, 1907	do	7	320,000
Be..	Aug. 6, 1906	Wagon..	18	1,380,000
	Sept. 5, 1906	Dairy...	8	166,000
	do	Wagon..	11	260,000
	July 6, 1907		22	34,000
	Aug. 8, 1907	Dairy..	20	23,600,000
	Aug. 20, 1907		17	94,000

Results of bacterial counts of market milk in Washington, 1906 and 1907—Continued.

Name.	Date.	Samples obtained at—	Temperature of milk when obtained (° C.).	Number of bacteria per cubic centimeter.
Bel	Aug. 13, 1906	Wagon	15	6,200,000
	Aug. 16, 1906	Dairy	12	3,600,000
	Sept. 10, 1906	Wagon	12	5,400,000
	Sept. 11, 1906	do	8	8,900,000
	Sept. 12, 1906	do	7	5,200,000
	Sept. 13, 1906	do	11	370,000
	Sept. 14, 1906	do	17	8,700,000
	Sept. 15, 1906	do	11	1,500,000
	Sept. 17, 1906	do	12	9,700,000
	Sept. 18, 1906	do	17	17,600,000
	Sept. 19, 1906	do	21	69,600,000
	Sept. 20, 1906	do	19	7,900,000
	Sept. 21, 1906	do	16	20,000,000
	July 6, 1907		12	2,300,000
	July 23, 1907	Wagon	21	3,520,000
	July 24, 1907	Dairy	8	14,400,000
	July 25, 1907	Wagon	18	730,000
	July 26, 1907	Children's Hospital	14	1,920,000
	do	do	14	2,000,000
	Aug. 1, 1907	Columbia Hospital	16	3,660,000
	Aug. 2, 1907	do	13	15 000,000
	Aug. 5, 1907	Wagon	11	8,000,000
	Aug. 19, 1907	do	11	3,000,000
Ben	July 17, 1907		21	4,300,000
Br	Aug. 7, 1907	Dairy	14.5	55,000,000
Bri	July 30, 1907		9	1,620,000
Bro	July 31, 1907	Wagon	20	150,000
	Aug. 7, 1907	do	18	113,000
Bu	July 5, 1907	do	18	51,000
Ca	July 11, 1907		20	16,300,000
	July 29, 1907	Dairy	9.5	20,000,000
Car	July 19, 1907		12	6,800,000
Ce	July 6, 1907		9	33,000,000
	July 23, 1907		9.5	30,800,000
	Aug. 6, 1907	Dairy	12.5	840,000
	Aug. 20, 1907	Wagon	19	6,000,000
C	July 5, 1907	Dairy	15	176,000
	July 16, 1907		13	101,000
	July 25, 1907	Dairy	15	800,000
	Aug. 13, 1907	do	16	4,000,000
	Aug. 30, 1907	do	12	70,000
Ch	Aug. 13, 1906	do	8	3
	Aug. 16, 1906	do	7	36,000
	Aug. 21, 1906	Wagon	9	17,000
	Aug. 22, 1906	do	8	190,000
	Aug. 23, 1906	do	11	64,000
	Aug. 24, 1906	do	11	42,000
	July 10, 1907		6.5	7,000
	July 17, 1907		9.5	140,000
	July 25, 1907		10	30,000
	July 29, 1907	Dairy	10	25,000
	Aug. 1, 1907	do	73.5	23,000
	Aug. 23, 1907	do		2,000

Results of bacterial counts of market milk in Washington, 1906 and 1907—Continued.

Name.	Date.	Samples obtained at—	Temperature of milk when obtained (° C.).	Number of bacteria per cubic centimeter.
Che	Aug. 13, 1906	Wagon	17	2,500,000
	Aug. 20, 1906	Dairy	15	44,000,000
	Aug. 28, 1906	do	12	33,000,000
	Sept. 11, 1906	Wagon	23	22,800,000
	Sept. 12, 1906	do	22	9,870,000
	Sept. 13, 1906	do	20	10,700,000
	Sept. 14, 1906	do	21	7,400,000
	Sept. 15, 1906	do	15	1,010,000
	Sept. 17, 1906	do	19	550,000
	Sept. 18, 1906	do	22	11,000,000
	Sept. 19, 1906	do	21	44,000,000
	Sept. 20, 1906	do	18	14,900,000
	Sept. 21, 1906	do	22	5,800,000
	July 16, 1907		21	30,800,000
	Aug. 2, 1907	Dairy	16	8,900,000
	Aug. 21, 1907	do	13	61,000,000
	Aug. 27, 1907	Wagon	14	1,900,000
	Aug. 30, 1907	do	8	21,000,000
Cla	July 19, 1907	do	14	208,000
	July 30, 1907	do	14	500,000
	Aug. 13, 1907	do	12	540,000
Cle	July 31, 1907	do	17	10,300,000
Clo	Sept. 6, 1906	Wagon	11	4,860,000
	July 16, 1907		14.5	113,000
	Aug. 2, 1907	Wagon	15	3,200,000
	Aug. 9, 1907	Dairy	16	1,600,000
	Aug. 21, 1907	do	15	4,000,000
	Aug. 30, 1907	Wagon	15	1,700,000
Cog	July 31, 1907		14	2,500,000
	Aug. 22, 1907	Dairy	9	8,900,000
	Aug. 26, 1907	do	13	2,100,000
Cor	Aug. 3, 1906	Wagon	21.5	12,500,000
	Aug. 9, 1907	do	19.5	160,000
Cud	July 15, 1907	do	13.5	2,280,000
	do	do	18	740,000
	Aug. 7, 1907	do	12	810,000
	Aug. 22, 1907	do	15.5	2,900,000
Du	Aug. 13, 1906	do	15	11,200,000
	Sept. 25, 1906	Dairy	9	380,000
	July 5, 1907	do	15	5,450,000
	July 17, 1907		8.5	65,400,000
	July 25, 1907		17	4,000,000
	Aug. 7, 1907	Dairy	19	116,000
	Aug. 29, 1907	do	12	710,000
Ec	Aug. 2, 1906	Wagon	21	5,900,000
Ed	July 19, 1907		14.5	1,180,000
	July 31, 1907		14	4,000,000
	Aug. 22, 1907	Dairy	11	221,000
Edw	July 8, 1907		22.5	33,000
	July 18, 1907	Wagon	24	420,000
	Aug. 22, 1907	do	23	600,000
En	July 30, 1907		19	2,500,000

I. of bacterial counts of market milk in Washington, 1906 and 1907—Continued.

Name	Date.	Samples obtained at	Temperature of milk when obtained (° C.).	Number of bacteria per cubic centimeter
Ev	Aug. 8, 1906	Wagon	15	9,200,000
	Aug. 10, 1906	do	16	45,900,000
	Aug. 13, 1906	do	15	15,600,000
	Aug. 14, 1906	do	15	18,200,000
	Aug. 15, 1906	do	15	8,400,000
	Aug. 16, 1906	do	17	2,500,000
	Aug. 17, 1906	do	16	6,800,000
	Aug. 20, 1906	do	23	19,200,000
	Aug. 22, 1906	Dairy	16	28,800,000
	July 9, 1907		5	5,700,000
	July 15, 1907		14	14,400,000
	July 24, 1907		16	16,000,000
	Aug. 12, 1907	Dairy	2	2,140,000
F	Aug. 20, 1906		13	2,400,000
	Aug. 21, 1906	Dairy	15	4,300,000
	Sept. 5, 1906	do	16	1,930,000
	July 5, 1907	do	15	1,320,000
	July 17, 1907		13.5	11,100,000
	July 25, 1907		11	1,600,000
	Aug. 9, 1907	Dairy	17	720,000
	Aug. 19, 1907	do	8	1,500,000
Fa	Aug. 22, 1906		14	1,640,000
	Sept. 25, 1906	Dairy	11	2,800,000
	July 9, 1907		13.5	74,000,000
	July 31, 1907	Wagon	18.5	416,000
	Aug. 15, 1907	Dairy	10.5	5,000,000
	Aug. 29, 1907	Wagon	15	2,000,000
Fr	Aug. 2, 1906	do	24	220,000
	Aug. 2, 1907	do	11	2,900,000
	Aug. 20, 1907	do	15	6,600,000
Fy	Aug. 22, 1907	do	19	11,500,000
Ga	July 10, 1907	do	20	10,300,000
Gi	Aug. 16, 1907	Dairy	18	170,000
Gl	Aug. 1, 1907	Wagon	10.5	1,320,000
	Aug. 6, 1907	do	17	123,000
	Aug. 19, 1907	do	13	700,000
	Aug. 27, 1907	do	8	2,100,000
Gle	Aug. 29, 1906	Wagon		1,655,000
	July 11, 1907		20.5	4,300,000
	July 15, 1907	Wagon	11.5	4,000,000
	July 29, 1907	Dairy	12	11,800,000
	Aug. 19, 1907	Wagon	14	10,000,000
	Aug. 26, 1907	Dairy	8.5	29,800,000
Go	July 13, 1907		11	930,000
	Aug. 6, 1907	Dairy	6	6,000
	Aug. 29, 1907	do	3	690,000

Results of bacterial counts of market milk in Washington, 1906 and 1907—Continued.

Name	Date	Samples obtained at	Temperature of milk when obtained (C.)	Number of bacteria per cubic centimeter
Gr	Aug. 14, 1906	Wagon	20	27,000,000
	Aug. 15, 1906	do	17	36,600,000
	Aug. 16, 1906	do	16	4,000,000
	Aug. 27, 1906	Dairy	15	11,300,000
	July 13, 1907		14	1,560,000
	July 18, 1907		13	2,170,000
	July 24, 1907		20	2,000,000
	July 26, 1907	Wagon	12	2,340,000
	Aug. 12, 1907	do	18.5	14,100,000
	Aug. 20, 1907	do	18	2,300,000
Gy	Aug. 15, 1907	Dairy	6	520,000
H	Aug. 6, 1906	Wagon	22	1,800,000
	Sept. 25, 1906	Dairy	11	2,500,000
	July 17, 1907			3,700,000
	Aug. 5, 1907	Dairy	6.5	1,020,000
	Aug. 27, 1907	Wagon	18	2,200,000
Ha	July 17, 1907	do	18	2,520,000
	Aug. 12, 1907	do	19	4,150,000
	Aug. 21, 1907	do	19	19,200,000
	do	Georgetown Hospital	12	50,000,000
	do	do	17	4,000,000
	Aug. 30, 1907	Wagon	14	4,000
Har	Aug. 28, 1906	Wagon	15	50,400,000
	Aug. 31, 1906	Dairy	12	43,100,000
	July 12, 1907		14	41,000,000
	Aug. 12, 1907	Dairy	16.5	115,000,000
	Aug. 23, 1907	do	9.5	14,200,000
He	Aug. 8, 1907	Wagon		17,000,000
	Aug. 23, 1907	do	8.5	3,600,000
Hi	Aug. 7, 1906	do	22	4,200,000
	Aug. 20, 1906	Dairy	23	5,300,000
	Aug. 21, 1906	do	18	6,600,000
	July 5, 1907	do	17	1,240,000
	July 25, 1907		23	3,280,000
	Aug. 27, 1907	Wagon	12	1,050,000
Hil	Aug. 3, 1906	do	24	42,000,000
	Aug. 2, 1907	do	21	243,000
Ho	July 12, 1907		9	2,100,000
	July 26, 1907	Dairy	13	1,040,000
	Aug. 16, 1907	do	8.5	4,300,000
	Aug. 28, 1907	Wagon	7.5	9,700,000
Hor	July 16, 1907	do	20	4,300,000
	Aug. 20, 1907	do	20	10,000
Hou	Aug. 9, 1907	do	23	560,000
	Aug. 30, 1907	do	19	36,800,000
Hy	July 25, 1907	do	24	7,000,000
	Aug. 12, 1907	do	22	12,000,000

Results of bacterial counts of market milk in Washington, 1906 and 1907—Continued.

Name.	Date.	Samples obtained at—	Temperature of milk when obtained (° C.).	Number of bacteria per cubic centimeter.
In	Aug. 13, 1906	Dairy	15.5	27,000,000
	Aug. 27, 1906	do	15	23,400,000
	Aug. 29, 1906	Wagon	10.5	30,000,000
	Aug. 30, 1906	do	12	53,400,000
	July 9, 1907		14	8,800,000
	July 22, 1907		12	1,710,000
	July 24, 1907		18	480,000
	July 29, 1907		18	1,190,000
	Aug. 7, 1907	Dairy	17	10,000,000
Ja	Aug. 12, 1907	Garfield Hospital	20	340,000
	do	do		10,000,000
Je	Aug. 13, 1906	Dairy	18.5	50,400,000
	Aug. 14, 1906	Wagon	19	40,600,000
	Aug. 15, 1906	do	21	5,700,000
	Aug. 16, 1906	do	15	30,600,000
	Aug. 17, 1906	do	19	4,800,000
	July 22, 1907		15	49,600,000
	Aug. 7, 1907	Dairy	14	4,900,000
	Aug. 23, 1907	do	9	1,990,000
Kl	Aug. 28, 1906	Wagon	16	65,820,000
	Sept. 11, 1906	do	14	40,800,000
	Sept. 12, 1906	do	16	3,900,000
	Sept. 13, 1906	do	18	1,415,000
	Sept. 14, 1906	do	18	24,600,000
	July 10, 1907		16.5	59,200,000
	July 26, 1907	Wagon	18	4,000,000
	Aug. 23, 1907	Dairy	9	3,800,000
La	Aug. 9, 1907	Wagon	16	3,800,000
Le	Aug. 22, 1906		22	2,400,000
	Aug. 28, 1906		17	5,000,000
	July 23, 1907	Wagon	21	7,200,000
	Aug. 21, 1907	do	20	1,500,000
Li	Aug. 16, 1906	Dairy	12	145,800,000
	Sept. 25, 1906	do	10	3,100,000
	July 6, 1907		16	1,050,000
	July 24, 1907		17	45,000,000
M.	Aug. 15, 1907	Dairy	19	28,000
Ma	July 29, 1907	Wagon	18	170,000
	Aug. 27, 1907	do	15	80,000
Mar	Aug. 14, 1907	do	18	2,300,000
Me	Aug. 9, 1907	do	25	16,200,000
Me	do	do	15	250,000
	Aug. 20, 1907	do	10	350,000
Mi	July 30, 1907	do	21	280,000,000
	Aug. 29, 1907	do	16	23,600,000
Mo	July 13, 1907		10	57,400,000
	Aug. 6, 1907	Dairy	12.5	31,000,000
	Aug. 15, 1907	do	9	27,000,000
	Aug. 28, 1907	do	8	4,200,000
Mor	Aug. 23, 1907	Wagon	12	520,000

Results of bacterial counts of market milk in Washington, 1906 and 1907—Continued.

Name.	Date.	Samples obtained at	Temperature of milk when obtained (° C.).	Number of bacteria per cubic centimeter.
Mou	Aug. 29, 1906	Wagon	15	550,000
	Sept. 26, 1906	Dairy	13	11,800,000
	July 18, 1907		14	42,000,000
	July 29, 1907	Dairy	8.5	100,000
	Aug. 26, 1907	do	4.5	1,420,000
Na	Aug. 8, 1906	Wagon	21	55,200,000
	Aug. 10, 1906	do	23	40,500,000
	Aug. 27, 1906	Dairy	18	43,800,000
	Sept. 8, 1906	Wagon	11	3,300,000
	Sept. 10, 1906	do	22	63,000,000
	Sept. 11, 1906	do	17	34,200,000
	Sept. 12, 1906	do	17	39,000,000
	Sept. 13, 1906	do	19	132,000,000
	Sept. 14, 1906	do	20	63,600,000
	July 10, 1907		13.5	510,000
	Aug. 9, 1907	Dairy	12	560,000
	Aug. 28, 1907	do	9	480,000
	Aug. 29, 1907	do	8	1,900,000
	Aug. 30, 1907	do	9	97,000
No	Sept. 5, 1906	Wagon	10	2,900,000
	Sept. 25, 1906	Dairy	12	660,000
O	July 15, 1907	Wagon	17.5	24,000,000
	Aug. 8, 1907	do	14.5	130,000
	Aug. 22, 1907	do	17	1,210,000
Ou	Aug. 30, 1906	Dairy	10	6,806,000
	Aug. 31, 1906	do	9	17,000,000
	Aug. 14, 1907	do	8	13,400,000
Ox	Aug. 6, 1907	Wagon	23.5	135,000
	Aug. 16, 1907	do	21	6,000
Pe	Aug. 16, 1906	Dairy	14	13,470,000
	Aug. 23, 1906	Wagon	14	6,700,000
	Aug. 29, 1906	do	18	7,000,000
	July 6, 1907		16.5	3,850,000
	July 16, 1907	Wagon	18.5	19,300,000
	July 23, 1907	do	21	28,000,000
	Aug. 8, 1907	Dairy	16	10,000,000
	Aug. 20, 1907		13	1,505,000
	Aug. 27, 1907	Wagon	11	34,600,000
Po	July 12, 1907		16	69,000,000
	Aug. 8, 1907	Dairy	10	440,000
	Aug. 19, 1907	do	11	44,000,000
Py	Aug. 5, 1907	Wagon	21	430,000
	Aug. 19, 1907	do	20	500,000
Qu	July 9, 1907		17	3,900,000
	July 29, 1907	Wagon	12	1,150,000
	Aug. 16, 1907		9	4,000,000
R	July 15, 1907		24	94,000
	Aug. 16, 1907	Wagon	23	8,000
Re	do.	do	25	1,000,000
Rei	Aug. 14, 1907	do	20	1,500,000
Ren	July 18, 1907		10.5	15,800,000
Ri	Aug. 3, 1906	Wagon	23.5	16,000,000

Results of bacterial counts of market milk in Washington, 1906 and 1907—Continued.

Name.	Date.	Samples obtained at—	Temperature of milk when obtained (° C.).	Number of bacteria per cubic centimeter.
Ro.	July 25, 1907	Wagon	21	330,000
	Aug. 12, 1907	do	21	370,000
Ru	July 12, 1907		12	320,000
	July 26, 1907		10.5	65,000,000
	Aug. 12, 1907	Dairy	11.5	21,400,000
	Aug. 15, 1907	Sibley Hospital	11	10,000,000
	Aug. 23, 1907	Dairy	10	1,550,000
	Aug. 29, 1907	Wagon	14	5,500,000
Sa	July 9, 1907		15.5	36,000,000
	July 22, 1907		9	5,300,000
	Aug. 5, 1907	Wagon	12	2,800,000
	Aug. 13, 1907	Dairy	11	50,000,000
	Aug. 20, 1907	do	9.5	3,700,000
	Aug. 28, 1907	do	11.5	7,000,000
	Aug. 29, 1907	do	5	15,500,000
	Aug. 30, 1907	do	9	101,000
S	July 30, 1907	Wagon	22	1,300,000
Sh	Aug. 2, 1906	do	24	60,000,000
	Aug. 20, 1906	do	24	307,800,000
	Aug. 21, 1906	do	23	80,000,000
	Aug. 22, 1906	do	24	4,000,000
	Aug. 23, 1906	do	24	1,000,000
	Aug. 24, 1906	do	19	9,800,000
	July 18, 1907		10	1,880,000
	July 31, 1907	Orphan Asylum	31	2,860,000
	Aug. 5, 1907	Wagon	15	480,000
	Aug. 7, 1907	do	18	2,440,000
	Aug. 13, 1907	Dairy	7	12,800,000
	Aug. 26, 1907	do	7	1,900,000
Sta	July 8, 1907		14	58,000,000
	Aug. 14, 1907	Dairy	8	5,000,000
	Aug. 28, 1907	do	4.5	1,400,000
She.	July 8, 1907		11	31,200,000
	Aug. 6, 1907	Dairy	10.5	1,410,000
	Aug. 15, 1907	do	7	182,000
	Aug. 28, 1907	do	6	700,000
Stan	July 11, 1907		11.2	4,000,000
	July 31, 1907	Dairy	8.2	23,000,000
	Aug. 26, 1907	do	7	440,000
St	Aug. 1, 1907	Wagon	24	75,000
	Aug. 19, 1907	do	20	700,000
	Aug. 27, 1907	do	20	2,000,000
Su.	Aug. 22, 1907	do	21	800,000
Stro	July 5, 1907	do	17	106,000
	July 19, 1907	do	21.5	2,030,000
Sp	Sept. 17, 1906	do	12	1,300,000
	Sept. 18, 1906	do	16.5	2,200,000
	Sept. 19, 1906	do	20	6,100,000
	Sept. 20, 1906	do	16	33,150,000
	Sept. 21, 1906	do	14.5	12,860,000
	July 13, 1907		16	4,270,000
	July 30, 1907	Wagon	14	4,700,000
	Aug. 15, 1907	Dairy	5	1,600,000

Results of bacterial counts of market milk in Washington, 1906 and 1907—Continued.

Name.	Date.	Samples obtained at	Temperature of milk when obtained (° C.).	Number of bacteria per cubic centimeter.
Spr	July 31, 1906	Wagon	20	1,600,000
	Aug. 6, 1906	do	20	9,580,000
	Aug. 24, 1906	Dairy	20	2,100,000
	Aug. 27, 1906	do	12	9,000,000
	July 17, 1907		23	250,000
	Aug. 5, 1907	Dairy	14	22,000,000
	Aug. 21, 1907	do	11	78,000,000
Spri	Aug. 3, 1906	Wagon	12	260,000
St	July 8, 1907		13	300,000
	July 26, 1907		10	940,000
	Aug. 6, 1907	Dairy	11	2,500,000
	Aug. 15, 1907	Wagon	14	200,000
	Aug. 23, 1907	Dairy	8.5	159,000,000
Su	Aug. 8, 1906	Wagon	22	44,400,000
	Aug. 14, 1907	do	15	30,000,000
Sw	Aug. 30, 1906	Dairy	9	14,000,000
	do	Wagon	8	11,500,000
	Aug. 31, 1906	Dairy	10	42,000,000
	do	Wagon	15	33,000,000
	July 12, 1907		21	2,570,000
	July 19, 1907		11	63,000,000
	July 22, 1907		9.5	87,500,000
	July 23, 1907		7	7,700,000
	Aug. 5, 1907	Wagon	16.5	650,000
	Aug. 8, 1907	do	13	2,000,000
	Aug. 14, 1907	Dairy	9	100,000,000
	Aug. 16, 1907	do	9.5	42,000,000
Ta	July 12, 1907		9.5	8,900,000
	Aug. 8, 1907	Dairy	15	2,050,000
	Aug. 15, 1907	do	13	100,000,000
Te	July 16, 1907	Wagon	22.5	640,000
	Aug. 2, 1907	do	21	400,000
Th	Aug. 14, 1906	do	17	1,300,000
	Aug. 15, 1906	do	15	2,400,000
	Aug. 16, 1906	do	24	4,800,000
	Aug. 17, 1906	do	25	12,000,000
	Aug. 24, 1906	do	24	15,600,000
	July 11, 1907		16	297,000
	July 18, 1907		11.5	57,000
	July 24, 1907	Wagon	18	1,080,000
	Aug. 13, 1907	Dairy	10	100,000
	Aug. 26, 1907	do	10	2,500,000
Tr	Sept. 26, 1906	do	13	420,000
	Aug. 2, 1907	do	16	1,190,000
	Aug. 13, 1907	do	23	180,000
	Aug. 21, 1907	do	20	4,200,000
Un	Aug. 23, 1906		12	13,400,000
	Aug. 27, 1906	Dairy	13	28,200,000
	July 8, 1907		13	109,000
	July 22, 1907		8	182,000
	Aug. 7, 1907	Dairy	11	107,000
	Aug. 16, 1907	do	9	5,000

Results of bacterial counts of market milk in Washington, 1906 and 1907—Continued.

Name	Date	Samples obtained at—	Temperature of milk when obtained (° C.).	Number of bacteria per cubic centimeter.
Va	Aug. 1, 1907	Wagon	24	1,000,000
Vi	Aug. 8, 1906	do	21	165,000,000
Wa	Aug. 1, 1906	do	18	400,000
	Aug. 2, 1906	do	18	3,200,000
	Aug. 3, 1906	do	19	520,000
	Aug. 6, 1906	do	22	200,000
	Aug. 7, 1906	do	21	290,000
	Aug. 8, 1906	do	12	156,000
	Aug. 9, 1906	do	15	40,000
	Aug. 10, 1906	do	15	55,000
	Aug. 11, 1906	do	16	41,000
	Aug. 13, 1906	do	14	48,000
	Aug. 14, 1906	do	14	39,000
	Aug. 15, 1906	do	14	80,000
	Aug. 16, 1906	do	19	130,000
	do	do	19	134,000
	Aug. 17, 1906	do	14	73,000
	Aug. 20, 1906	do	23	108,000
	Aug. 21, 1906	do	18	196,000
	Aug. 28, 1906	do	16	86,000
	do	do	16	84,000
	Sept. 26, 1906	Dairy	10	2,100,000
	July 19, 1907		9	3,080,000
	July 24, 1907	Wagon	10.5	340,000
	Aug. 1, 1907	Dairy	4.5	210,000
	Aug. 13, 1907		5.5	160,000
Wal	Aug. 20, 1906	Wagon	22	28,800,000
	Aug. 21, 1906	do	24	238,000,000
	Aug. 22, 1906	do	23	63,000,000
	Aug. 23, 1906	do	23	119,000,000
	Aug. 24, 1906	do	22	201,000,000
	July 23, 1907		5	70,500,000
	Aug. 14, 1907	Dairy	11	26,000,000
	Aug. 28, 1907	do	21	2,800,000
	Aug. 29, 1907	do	8	2,000,000
Way	Aug. 2, 1906	Wagon	24	31,800,000
Wy	July 15, 1907		15	7,000,000
	July 19, 1907		12	6,250,000
	Aug. 21, 1907	Wagon	18	6,800,000
	Aug. 26, 1907	do	9	590,000
	Sept. 4, 1907	do	17	3,900,000
	Sept. 5, 1907	do	4	10,300,000
Average for 1906			16.5° C.	22,134,000
Average for 1907			14.2° C.	11,270,000

BACTERIAL COUNTS IN OTHER CITIES.

The great bacterial contamination of milk in other cities is given in order to compare with the results found in Washington.

The statement is frequently made that the milk of American cities has fewer bacteria than that of European cities. The methods used for making bacterial counts differ, so that comparisons are difficult to make. The larger cities have a much greater bacterial contamination in their general milk supply than the smaller cities and towns. We would expect this difference when we recall how much easier it is to obtain milk less than 24 hours old in villages and small towns.

In comparing the following figures it must be remembered that in some instances the milk is collected as it reaches the city, while in other instances, corresponding to our work in Washington, the samples were taken as they reached the consumer. Bacteria multiply enormously between the time the milk arrives in a city and the time it is delivered to the consumer.

Von Geuns[a] in 1885 was the first to give us information concerning the number of bacteria contained in milk. He found 10,545 bacteria per cubic centimeter in the so-called pasteurized milk sold in Amsterdam.

Clauss,[b] 1889, made eight examinations of the fresh dairy milk of Würzburg in the winter of 1888–89 and found the average bacterial content to be between 1,000,000 and 2,000,000, the lowest count being 222,000 and the highest 2,300,000. The author says that Hohenkamp[c] in the summer, in Würzburg, found the bacterial content to range between 1,900,000 and 7,200,000 per cubic centimeter.

Cnof,[d] 1889, working with Escherich, found in the milk of Munich, as it came to the hands of the consumer five to six hours after the milking, that the bacterial content ranged between 200,000 and 6,000,000 per cubic centimeter; a few moments after milking the number ranged between 60,000 and 100,000 per cubic centimeter.

Renk,[e] 1891, found between 6,000,000 and 30,700,000 bacteria per cubic centimeter in the milk supply of Halle. Further, from a series of 30 tests it was found that an average of 15 milligrams of cow's excrement per liter was contained in the milk supply of Halle; Leipzig, 3.8 milligrams; Berlin, 10.3 milligrams, and Munich, 9 milligrams.

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[a] Von Geuns, J.: Ueber die Einwirkung des sog. "Pasteurisirens" auf die Milch. Arch. f. Hyg., vol. 3, 1885, p. 464.

[b] Clauss, Johannes: Bacteriologische Untersuchungen der Milch im Winter 1888–89 in Würzburg mit besonderer Berucksichtigung der Milchsäure Bildenden Bacterien. Inaug. disserta., Würzburg, 1889. 38 p. 8°.

[c] Hohenkamp: Arch. f. Hyg., vol. 14, p. 260.

[d] Cnof: Quantitative Spaltpilzuntersuchungen in der Kuhmilch. Cent. f. Bakt., vol. 20, 1889, p. 553.

[e] Renk: Ueber Marktmilch in Halle. Munch. med. Woch. 1891. Rev. in Escherich, Cent. f. Bakt., vol. 10, 1891, p. 193.

Uhl[a] in 1892 showed the great fluctuation in the bacterial content of the milk of Giessen. In May, 1892, from 30 examinations he found it ranged between 83,000 and 169,600,000; in June the lowest count was 10,500 and the highest 13,600,000. The average in May was 22,900,000, and the average in June was only 2,900,000.

Sedgwick and Batchelder,[b] 1892, were the first to record the bacterial content of milk from an American city. From a series of examinations of the milk supply of Boston and its suburbs they report as follows:

City.	Samples.	Bacteria per cubic centimeter.	City.	Samples.	Bacteria per cubic centimeter.
Charlestown	8	4,222,500	Roxbury	17	1,874,300
South Boston	9	2,778,000	North End	[illegible]	708,100
Jamaica Plain	10	3,259,600	Back Bay	7	1,189,800

These samples were taken directly from the milk wagons and planted at once.

From groceries 16 samples additional were taken which averaged 4,577,000 bacteria per cubic centimeter.

Ten samples collected from well-to-do families averaged 1,438,000 bacteria per cubic centimeter.

Forty-four samples of the so-called "railroad" milk from one dealer averaged 500,000 per cubic centimeter. The extremes were 5,664,000 and 2,200.

Another set of ten samples examined on arrival in Boston averaged 371,000 per cubic centimeter.

Knochenstiern[c] in 1893 gives the results of examination of milk at Dorpat, Russia, between September 18 and January 25, as follows:

	Average bacteria per cubic centimeter.
From milkmen	10,200,000
Village milk	12,000,000
Market milk	25,000,000
Shop milk	30,000,000

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[a] Uhl. Untersuchungen der Marktmilch in Giessen. Zeit. f. Hyg., vol. 12, 1892, p. 475.

[b] Sedgwick and Batchelder. A bacteriological examination of the Boston milk supply. Boston Med. and Surg. Journ., vol. 126, 1892, p. 25.

[c] Knochenstiern, Hugo. Ueber dem Keimgehalt der Dorpater Marktmilch nebst einigen bacteriologischen Untersuchungen von Frauenmilch. Inaug. Disserta., Dorpat. 1893. 51 p. 8°.

The same author gives abstracts of findings by the following authors:

Author	City.	Average bacteria per cubic centimeter.
Bujwid (16 examinations)	Warsaw	4,000,000
Geuns (dairy milk)	Amsterdam	2,500,000
Cunningham, D. (1891)	Calcutta	3,400 to 3,000,000

Rowland,[a] 1895, found the average bacterial content of 25 samples of milk in London to be 500,000 per cubic centimeters.

Frye[b] in 1896 examined nine samples of milk in Buffalo as it was delivered to the consumer from December 28, 1895, to June 11, 1896, and found the bacterial content to range from 48,000 to 43,600,000 per cubic centimeter.

In six samples of grocery milk examined from January 26 to June 11, 1896, the bacterial content ranged between 25,000 and 25,000,000 per cubic centimeter.

In seven samples of "certified milk" the bacterial content ranged between 4,400 and 132,700 per cubic centimeter.

Pakes[c] in 1900 states that London's milk supply contains between 3,000,000 and 4,000,000 bacteria per cubic centimeter.

Park,[d] 1901, found the milk in New York City to contain, as a rule, excessive numbers of bacteria. During the coldest weather the milk in the shops averages over 300,000 bacteria per cubic centimeter, during cool weather about 1,000,000, and during the hot weather about 5,000,000. He found the condition of the average city milk very different, depending upon temperature and other conditions. The milk as it is received in New York from the railroads averages over 5,000,000 bacteria per cubic centimeter, the lowest count being 52,000 and the highest 35,200,000.

Burrage[e] in 1901 states that American cities appear to have better milk from a bacterial standpoint than European cities. In the latter, milk seldom contains less than 5,000,000 bacteria per cubic centimeter. In the milk supply of Middletown, Conn., the number of bacteria was found to be comparatively low, the milk being delivered to the consumer within a few hours after milking. The bacteria varied from 11,000 to 300,000 per cubic centimeter.

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[a] Rowland, Sidney D. Report of 25 samples of milk examined as to their bacterial flora. Brit. Med. Journ., 1895, vol. 2, p. 321.

[b] Frye, Maud J. Notes upon the estimation of the number of bacteria in milk. Med. Rec., 1896, vol. 2, p. 442.

[c] Pakes, Walter. The application of bacteriology to public health. Lancet, 1900, vol. 1, p. 311.

[d] Park, W. H. The great bacterial contamination of the milk of cities. Can it be lessened by the action of health authorities? Journ. Hyg., vol. 1, 1901, p. 391.

[e] Burrage, Severance: Some sanitary aspects of milk supplies and dairying. Iowa Board Health, Eleventh Bien. Rep., 1901, p. 575.

Goler,[a] 1903, states that prior to 1900 the average bacterial counts of 86 samples of Rochester, N. Y., milk showed 837,000 per cubic centimeter, excluding 26 per cent of the samples which contained over 5,000,000 bacteria per cubic centimeter.

Dodd,[b] 1904, gives the following:

District.	Standard of shop.	Average bacteria per cubic centimeter	District	Standard of shop.	Average bacteria per cubic centimeter
City of London......	Good class.	1,800,000	Islington...	Good class....	1,600,000
City of Westminster...	...do...	1,600,000	Finsbury. ...	....do.......	2,300,000
Holborn....... ...	...do.....	4,800,000	Do......	Poor class....	3,200,000

Byrnes,[c] 1904, speaking of milk inspection in Philadelphia, says: "Another branch of this subject is the almost incredible number of bacteria found in our milk supply, which vary from 1,600 to 21,000,000 per cubic centimeter."

Jordan,[d] 1904, found that the market milk of Chicago contained an average of 9,361,000 bacteria per cubic centimeter in April, 10,071,000 in May, and 18,924,000 in June, 1904. Sixteen per cent of the samples examined contained over 20,000,000 bacteria per cubic centimeter.

Bergey,[e] 1904, found as a result of the examination of ten samples taken at random from a large series of examinations made in July, 1900, from milk taken at railroad depots in Philadelphia, an average bacterial content of 4,802,355 per cubic centimeter. The author gives a table showing the reported average bacterial content per cubic centimeter of the milk in other American cities, as follows:

	Bacteria per cubic centimeter.
New York..	4,000,000
Boston..	2,300,000
Chicago..	2,350,000
Baltimore..	4,000,000
Wilmington..	7,000,000

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[a] Goler, George W.: The influence of the municipal milk supply upon the deaths of young children. N. Y. State Journ. Med., vol. 3, 1903, p. 493.

[b] Dodd, F. Lawson: The problem of the milk supply. London, 1904. 77 p. 8°.

[c] Byrnes, W. J.: Annual report of the chief inspector of milk for the year 1903. Philadelphia Bur. Health, Ann. Rep., 1903, p. 76.

[d] Analyses of Chicago Market Milk, a report by the health and sanitation committee of the Civic Federation of Chicago, 1904.

[e] Bergey, D. H.: Sanitary supervision of the collection and marketing of milk. Univ. Pa. Med. Bull., vol. 17, 1904, p. 187.

He says that European market milk has been found to contain a greater average bacterial count, ranging from 5,000,000 to 10,000,000, and frequently 20,000,000 to 180,000,000.

Proskauer, Seligmann, and Croner,[a] 1907, found that Danish milk sold in Berlin in the summer varied, in round numbers, between 5,000,000 bacteria per cubic centimeter and innumerable quantities. In the winter this milk contained about 2,140,000 bacteria per cubic centimeter.

The same investigators found that the market milk of Berlin averaged 3,500,000 bacteria per cubic centimeter in summer and 567,000 in winter.

Knox and Schorer,[b] 1907, state that several summers ago the quality of the milk supplied to the working classes in Baltimore was studied during two successive summers at the laboratory of the Thomas Wilson Sanitarium. Much of the milk on sale at the small stores was shown to be unfit for consumption, having a high bacterial count.

It will thus be seen that the general market milk of Washington, as well as that of other large American and European cities, is worthy the serious attention of health officers so far as excessive bacterial contamination is concerned.

Addenda. At the last meeting of the American Association of Medical Milk Commissions the following report of the committee on bacteriological standards for certified milk was adopted:

The methods, so far as applicable, shall be those recommended by the committee on standard methods of bacteriological milk analysis of the laboratory section of the American Public Health Association.

Bacterial counts for certified milk should be made at least once a week.

Use agar-agar made according to the recommendation of the committee of the American Public Health Association containing 1.5 per cent agar and a reaction of +1.0 to phenolphthalein.

Grow at 37° C. for forty-eight hours, or at 22° C. for five days, or 27° C. for three days.

When in bottles, milk samples should be obtained in original packages and brought direct to the laboratory unopened.

As soon as practicable upon arrival at the laboratory, open the bottle with aseptic precautions and thoroughly mix the milk either by pouring back and forth between the original bottle and a sterile bottle, or by agitation for two minutes.

Make no less than two plates for each sample.

Make a control of each lot of medium and apparatus at each testing.

Dilute the milk in the proportion of 1 part of milk to 99 parts of sterile water; shake 25 times and plate 1 cubic centimeter of the dilution.

Express results in multiples of the dilution factor.

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[a] Proskauer, B., Seligmann, E., and Croner, Fr.: Über die Beschaffenheit der in Berlin eingeführten dänischen Milch. Ein Beitrag zur hygienischen Milchkontrolle. Zeit. f. Hyg., vol. 57, 1907, p. 173–247.

[b] Knox, J. H. Mason, and Schorer, Edwin H.: A study of hospital and dispensary milk in warm weather; with special reference to pasteurization. Arch. Pediatrics, July, 1907.

12. THE GERMICIDAL PROPERTY OF MILK.

THE GERMICIDAL PROPERTY OF MILK.

By Milton J. Rosenau, *Director Hygienic Laboratory, Public Health and Marine-Hospital Service*; and George W. McCoy, *Passed Assistant Surgeon, Public Health and Marine-Hospital Service.*

INTRODUCTION.

Judged by the number of colonies that develop upon agar plates, the bacteria in milk first diminish then increase in number. This occurs only in raw milk during the first few hours after it is drawn. Although the bacteria seemingly decrease in number, they never disappear entirely. After this initial decrease there is a continuous and rapid increase until the milk contains enormous numbers.

It was early noted that under certain conditions raw milk may keep longer than heated milk. In other words, the property of milk to restrain the growth of bacteria is destroyed by heat.

Before this so-called "germicidal property of milk" was discovered it had been observed that fresh blood, or blood serum, had distinct powers of destroying bacteria. Further, that blood resists putrefactive and fermentative changes. It is now well known that blood, apart from the phagocytic action of its cells, has definite germicidal properties. This is due to substances in solution in the blood serum which have the power of clumping, killing, or dissolving the bacterial cells. This power of the blood is an important protection against bacterial invasion. Similar uses have been assigned to the "antibodies" in milk. The germicidal properties of blood are destroyed by heat and disappear spontaneously in a short time after it has been removed from the body.

Not only the blood, but other body fluids have germicidal properties in varying degree, so that it was not surprising when similar powers were attributed to milk, especially when we consider that the fluid part of milk, with many of its constituents, is secreted directly from the blood.

This initial power of milk to destroy bacteria or to restrain their multiplication is feeble and variable. The germicidal properties of milk have been much misunderstood, especially by dairymen, some of whom insist that advantage may be taken of this property for the preservation of milk without the use of ice.

When we stop to consider that bacteria frequently enter the imperfectly closed orifice of the teat and grow in the milk contained in the milk cisterns, and that they often invade the finer tubules of the gland structure where the milk is being freshly secreted, we must be convinced that the "germicidal" power of milk must be exceedingly feeble, if it exist at all.

This property varies with the milk of different animals, and also in the milk of the same animal at different times.

There is evidence to show that the restraining action of fresh raw milk upon the growth of bacteria varies with the bacterial species, and when we inquire into the causes of the phenomenon we find that this is what we might expect.

When micro-organisms are transferred to a strange medium they sometimes hesitate, until they become sufficiently accustomed to the new surroundings, before they begin to multiply. Our experiments show that this is by no means always the case and can not account for the facts now under consideration.

We know that the serum of milk may contain "antibodies" in appreciable and variable quantity similar to those found in the blood. For instance, diphtheria, tetanus, and other antitoxins have been demonstrated in the milk of immunized animals. We might also expect small quantities of the agglutinating, bactericidal, and bacteriolytic substances present in blood serum to pass into the milk serum. Agglutinins in milk would apparently diminish the number of bacteria contained therein when estimated by the number of colonies that develop on agar plates. This might occur even though the number of bacteria present were actually increased. Microscopic examination of the bacteria in milk made at once after milking, and again in eight hours, demonstrates that such agglutination actually takes place. This is confirmed by our other technique.

We know that milk always contains large numbers of leucocytes many of them of the polymorphonuclear variety. These are known to be phagocytes, and we might assume that they are active in milk for a short time after it is drawn. In fact we have found that some of the leucocytes actually contain more bacteria after eight hours than when the milk is freshly drawn.

If phagocytosis played a part in the diminution in the number of bacteria in milk, we must assume that the milk serum has opsonic power.[a] Our work shows that phagocytosis plays no essential rôle in the apparent reduction in the number of bacteria in fresh milk.

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[a] In fact Woodhead and Mitchell claim to have demonstrated opsonins in milk. Journ. of Path. and Bactr., vol. 11, 1906-7, p. 408.

It seems likely that the germicidal property of milk corresponds to a similar property of fresh blood serum. This makes it probable that the causes are numerous and complex. Further, it explains why the action is variable in different milks and in milk from the same animal at different times. It also gives us a clew as to why the action is to a certain extent specific.

Although the germicidal property of fresh milk is feeble, it must be of value to the suckling. This self-evident fact emphasizes the importance of using fresh milk for artificial feeding.

EXAMPLES OF THE GERMICIDAL ACTION.

The following examples show that milk when fresh and raw actually restrains the growth of bacteria as judged by the development of colonies upon agar plates. Whether the bacteria are restrained, destroyed, or clumped is not evident from such technique.

This series shows the effect upon the growth of the bacteria that usually contaminate milk. These results show a restraining, rather than a germicidal action, which varies with the temperature. Actual reduction in numbers is more apparent from a study of our work with pure cultures (*vide infra*).

Table No. 1.—*Milk from a healthy cow (No. 1).*

[Immediately after milking contained 400 bacteria per cubic centimeter.]

Time after milking	Bacteria per cubic centimeter at different temperatures.		
	Room temperature, 16° to 23° C.	15° C.	37° C.
2 hours	430	...	...
4 hours	100	450	350
6 hours	350	600	2,100
8 hours	150	300	545,000
10 hours	500	250	1,780,000
12 hours	400	300	32,800,000
14 hours	500	400	75,500,000
24 hours	5,000	2,000	Sour.
36 hours	60,000	2,000	
48 hours	366,000	1,000	...
60 hours	780,000	3,800	
72 hours	24,200,000	61,000	
84 hours	250,000,000	118,000	
96 hours	330,000,000	3,080,000	
108 hours	910,000,000	33,400,000	
120 hours	Sour.	192,000,000	
144 hours	...	Innumerable	

TABLE No. 2.—*Milk from a healthy cow (No. 1).*

[Immediately after milking contained 500 bacteria per cubic centimeter.]

Time after milking.	Bacteria per cubic centimeter at different temperatures.		
	Room temperature, 26° to 29° C.	15° C.	37° C.
2 hours	1,300		
4 hours	700	900	11,300
6 hours	400	500	38,000
8 hours	7,800	600	342,000
10 hours	29,000	1,200	50,000,000
24 hours	340,000,000	80,000	Sour.
48 hours	Innumerable.	1,380,000	
72 hours	Sour.	89,000,000	
96 hours		Sour.	

TABLE No. 3.—*Milk from a healthy cow (No. 1).*

[Immediately after milking contained 8,300 bacteria per cubic centimeter.]

Time after milking.	Bacteria per cubic centimeter at various temperatures.		
	Room, 26° to 29° C.	15° C.	37° C.
2 hours	8,000		
3 hours	2,000	2,000	2,000
4 hours	6,000		
5 hours		2,000	
6 hours	2,000	1,000	6,000
7 hours	1,000		20,000
8 hours	1,000	5,000	166,000

THE EFFECT OF TEMPERATURE.

Temperature has a decided influence upon this phenomenon. When the milk is kept warm (37° C.) the decrease in the number of colonies is striking, but of short duration. When the milk is kept cool (15° C.) the decrease is less marked, but more prolonged. This is well illustrated by the curves, Figs. 9 to 14.

These curves were plotted from the following tables, which show the germicidal properties of milk for individual species of bacteria. The experiments were conducted as follows:

Milk was obtained from a healthy cow, using particular care to prevent outside contamination. For this milk we are greatly indebted to Doctor Schroeder and Mr. Cotton, of the Experiment Station, Department of Agriculture, Bethesda, Md.

In addition to the usual precautions, a cloth wet with bichloride solution was placed under the cow, permitting only the teats to proect through. The foremilk was discarded and about 10 to 15 cubic centimeters of the midmilk was introduced directly into sterile test tubes. Some of these tubes, tested soon afterwards, were found to be practically sterile; other contained about 60 bacteria per cubic centimeter. Without delay, a loopful of a pure culture of the organism to be tested from a 24-hour-old agar slant was placed in the tubes of the freshly drawn milk. Two of the tubes were contaminated with each culture tested; one of them was kept at 15° C. and the other at 37° C.

For a control, a similar loopful of culture was planted in a tube of whole milk that had been sterilized fractionally upon three successive days. At the intervals shown in the tables the tubes were shaken in order to obtain a uniform suspension, and a loopful planted upon agar by the plate method. Duplicate plates showed that the loop always took up about the same quantities. The results, however, do not pretend to mathematical accuracy, but are sufficiently consistent to show any marked increase or diminution in the number of colonies.

TABLE No. 4.—*B. typhosus.*

Milk from healthy cow (No. 2).	Number of bacteria per loop—			
	At 15° C.		At 37° C.	
	In raw milk.	In sterilized milk.	In raw milk.	In sterilized milk.
At once, after milking	6,720	10,400	6,580	4,860
2 hours later	6,100	10,180	4,300	6,600
4 hours later	5,940	15,000	985	15,360
6 hours later	7,920	20,000	388	(a)
8 hours later	1,860	20,000	62	(a)
10 hours later	4,620	11,000	180	(a)
12 hours later	3,180	37,500	1,800	(a)
24 hours later	4,200	31,000	(a)	(a)
48 hours later	(a)	(a)	...	...

[a] Innumerable.

Here it was plain that there was an actual diminution in the number of typhoid colonies from the tube kept at 37° C. during the first eight hours, after which multiplication began. The bacteria in the sterilized milk used as a control increased almost from the start. At 15° C. the restraining effect is similar, but less pronounced and more prolonged.

TABLE No. 5. *B. dysenteriæ.*

Milk from healthy cow (No. 2).	Number of bacteria per loop—			
	At 15° C.		At 37° C.	
	In raw milk.	In sterilized milk.	In raw milk.	In sterilized milk.
At once, after milking	2,820	3,960	4,680	27,000
2 hours later	1,380	3,800	1,000	15,000
4 hours later	900	4,200	720	27,000
6 hours later	756	4,400	540	(a)
8 hours later	890	6,360	348	(a)
10 hours later	660	8,040	1,296	(a)
12 hours later	540	9,600	7,200	(a)
24 hours later	121	15,000	15,000	(a)
48 hours later	109	a	a	....
72 hours later	50	....	....	....
96 hours later	55	....	....	....

a Innumerable.

This shows the same phenomenon as in the case of the typhoid bacillus, excepting that the restraining power of the raw milk at 15° C. is more marked and prolonged for the dysentery bacillus.

TABLE No. 6. *B. lactis aerogenes.*

Milk from healthy cow (No. 2).	Number of bacteria per loop—			
	At 15° C.		At 37° C.	
	In raw milk.	In sterilized milk.	In raw milk.	In sterilized milk.
At once, after milking	19,920	8,500	16,000	23,000
2 hours later	14,220	9,600	24,000	57,000
4 hours later	15,120	6,800	7,750	(a)
6 hours later	16,680	7,560	1,200	....
8 hours later	10,980	10,000	418	....
10 hours later	12,450	10,000	1,335	....
12 hours later	9,720	(a)	1,500	....
24 hours later	9,440	(a)	(a)	....
48 hours later	a	....	....	....

a Innumerable.

This organism is one of the common causes of lactic acid fermentation. It is practically always present in milk unless drawn with extraordinary precautions. It is evident that this particular sample of raw milk exerted the same temporary restraining influence upon this organism that it did upon the pathogenic species.

TABLE NO. 7.—*V. choleræ.*

Milk from healthy cow (No. 2).	Number of bacteria per loop—			
	At 15° C.		At 37° C.	
	In raw milk.	In sterilized milk.	In raw milk.	In sterilized milk.
At once, after milking	2,820	3,540	6,000	8,460
2 hours later	740	7,860	6,720	9,780
4 hours later	1,440	4,980	987	15,795
6 hours later	1,740	5,100	1,711	[a]
8 hours later	1,800	5,280	6,300	[a]
10 hours later	820	7,200	11,880	[a]
12 hours later	1,010	9,300	15,660	[a]
24 hours later	1,440	16,000	[a]	[a]
48 hours later	900	[a]		
72 hours later	6,700			
96 hours later	3,000			

[a] Innumerable.

The milk had practically the same power of restraining the cholera vibrio that it had for the other bacteria tested.

TABLE NO. 8.—*B. diphtheriæ.*

Milk from healthy cow (No. 2).	Number of bacteria per loop—			
	At 15° C.		At 37° C.	
	In raw milk.	In sterilized milk.	In raw milk.	In sterilized milk.
At once, after milking	270	160	470	170
2 hours later	30	230	333	1,180
4 hours later	230	430	285	Lost.
6 hours later	270	160	265	370
8 hours later	330	300	275	960
10 hours later	600	120	150	2,700
12 hours later	200	180	215	3,800
24 hours later	145	215		8,420
48 hours later	..	..		[a]

[a] Innumerable.

The results obtained with the diphtheria bacillus are perhaps not quite so graphic as with the other organisms, partly for the reason that the diphtheria bacillus is not motile and it is difficult to obtain a uniform suspension; and also partly for the reason that it grows so slowly, if at all, at 15° C. However, at 37° C. definite restraining action is evident in the raw milk as compared with the sterilized milk.

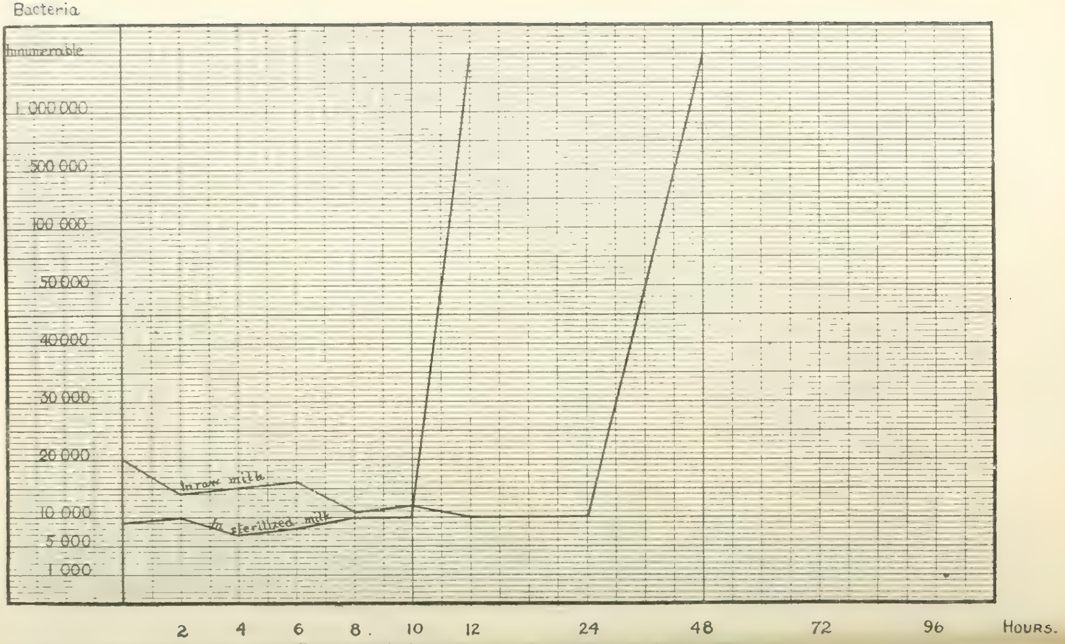

Fig. 9.—Growth of *B. lactis aerogenes* in raw and sterilized milk at 15° C.

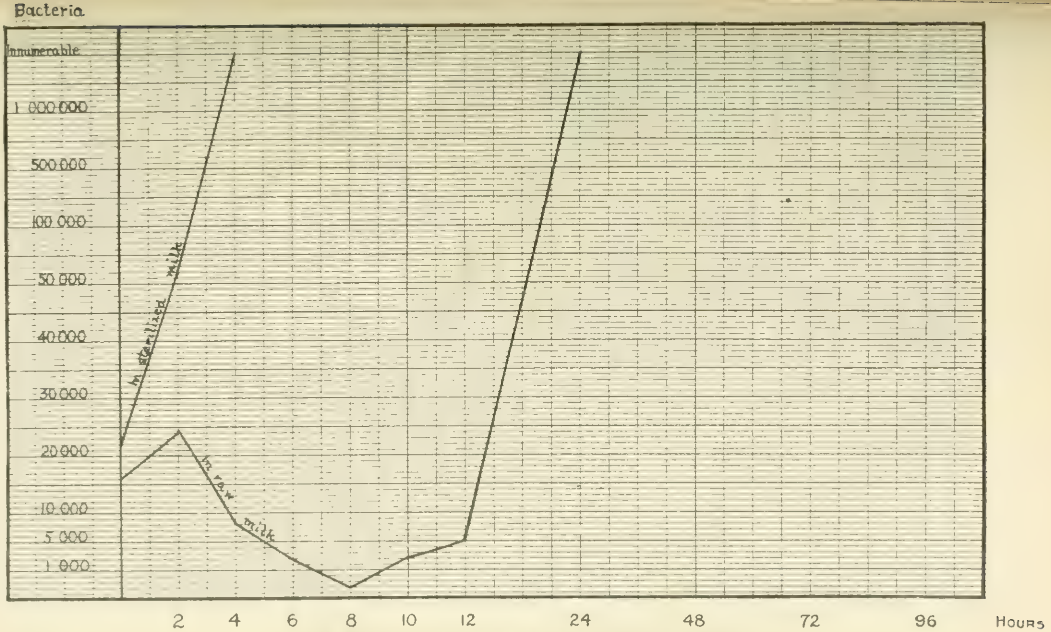

Fig. 10.—Growth of *B. lactis ærogenes* in raw and sterilized milk at 37° C.

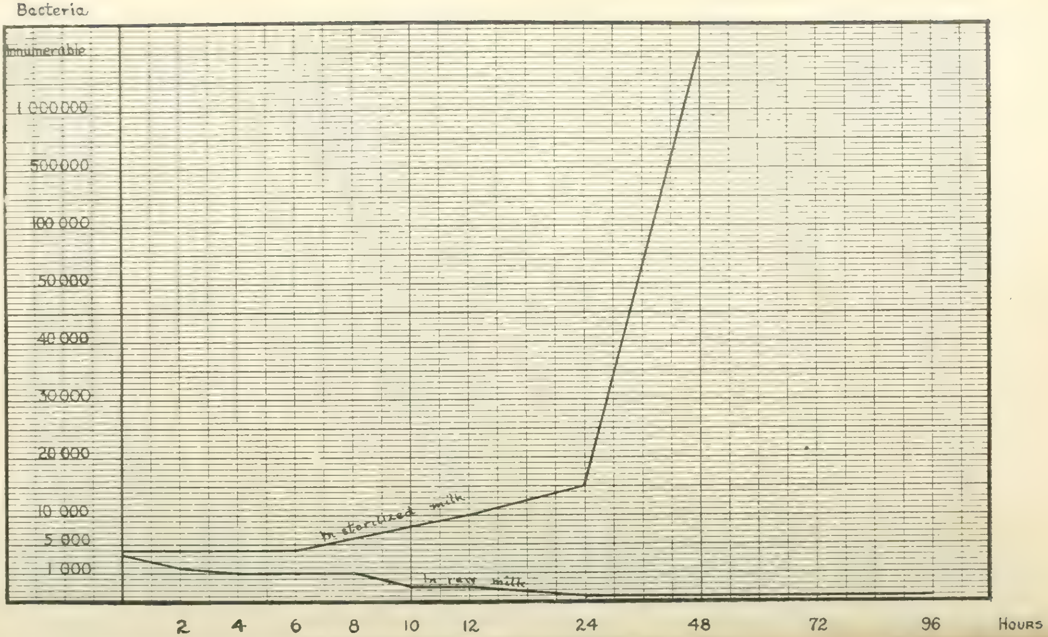

FIG. 11.—Growth of *B. dysenteriæ* in raw and sterilized milk at 15° C.

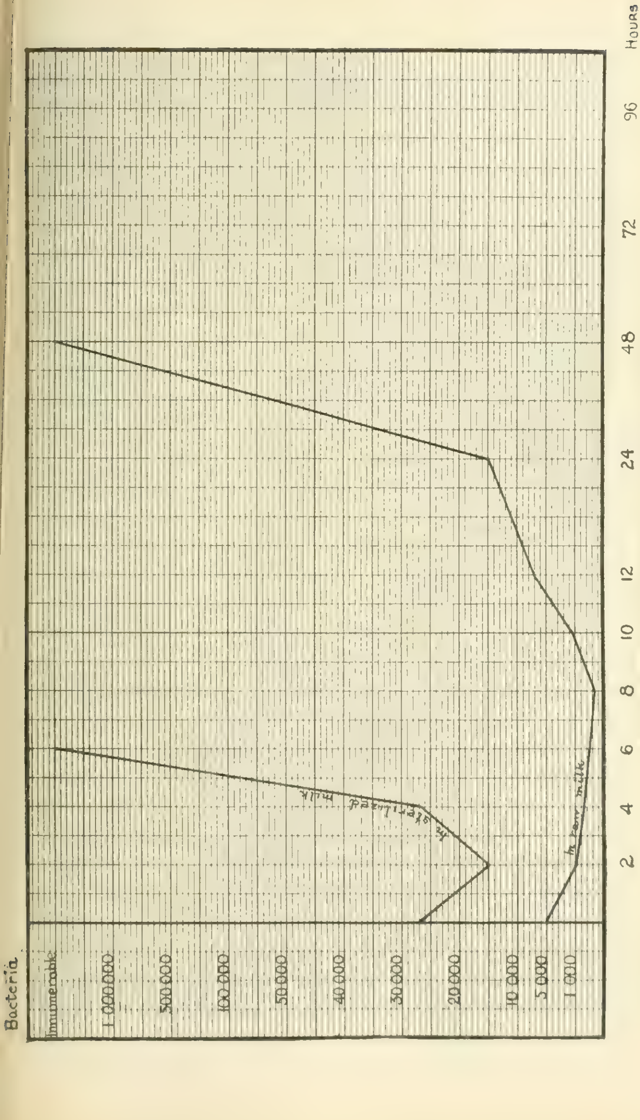

Fig. 12.—Growth of *B. dysenteriae* in raw and sterilized milk at 37° C.

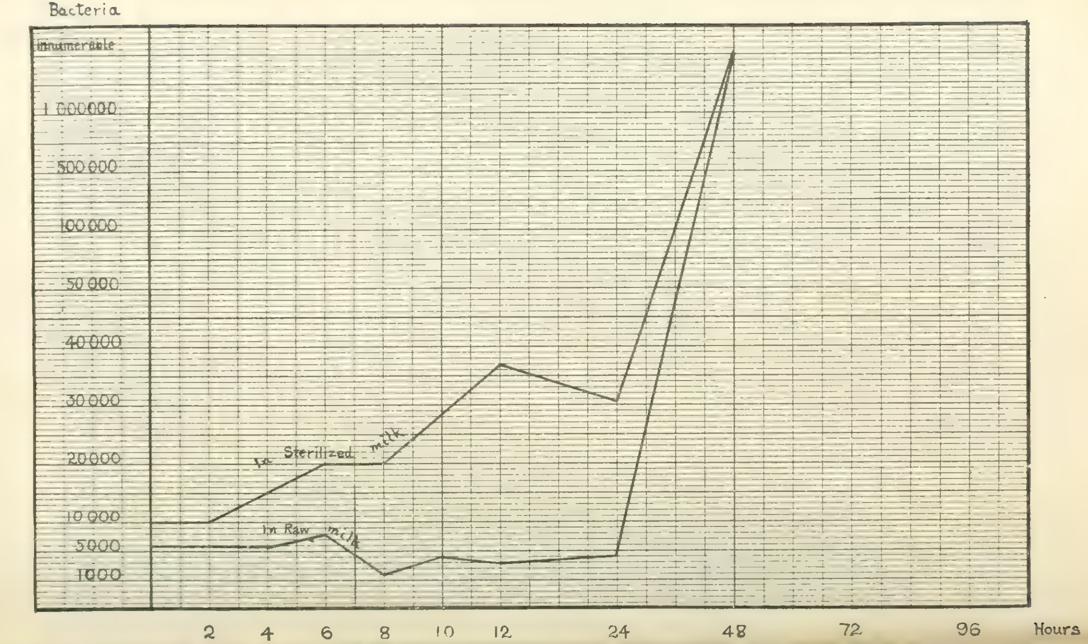

FIG. 13.—Growth of *B. typhosus* in raw and sterilized milk at 15° C.

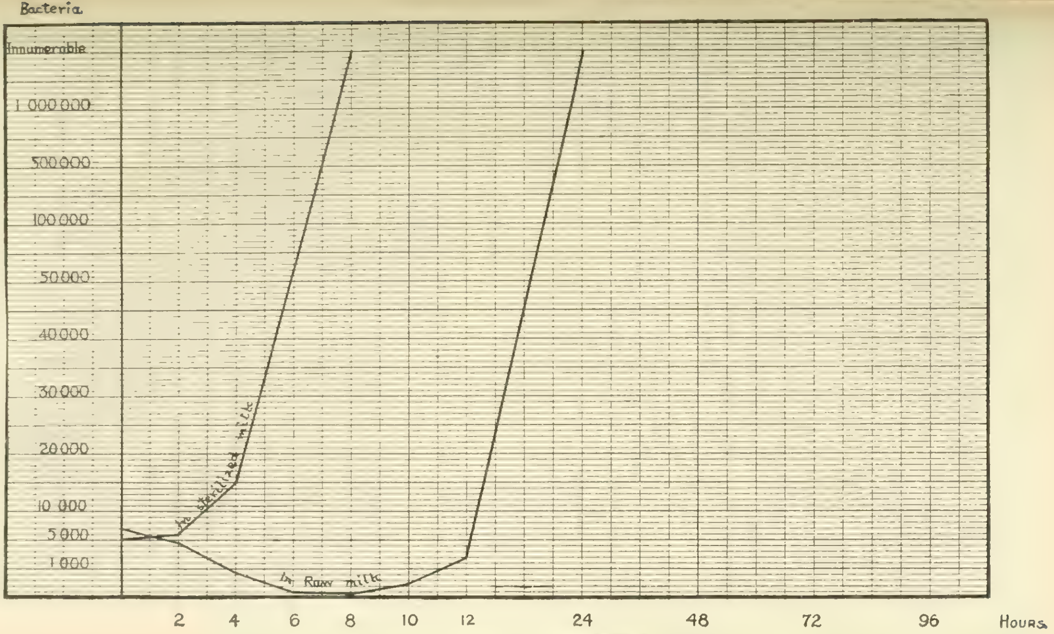

FIG. 11.—Growth of *B. typhosus* in raw and sterilized milk at 37° C.

RELATION TO AGGLUTINATION.

Agglutination of bacteria in milk is somewhat difficult to demonstrate macroscopically. Under the microscope, in stained preparations, the bacteria are in large and dense clusters in raw milk after standing eight hours at 37° C. In the boiled milk used as a control the bacteria under the same conditions appear singly or in very short chains or small clumps. In our work care was taken to break up all clumps in the suspensions used to inoculate both the raw and the boiled milk.

Vigorous shaking also gave results that plainly proved that agglutination is one of the factors that cause an apparent decrease in the number of bacteria.

The agglutinated bacterial clusters were broken asunder by one or both of the following methods, stated in each table:

1. Vigorous shaking of the milk for about five minutes in a glass-stoppered cylinder.

2. Drawing the suspension in and out a number of times through a capillary pipette, the end of which is broken off square and closely applied to the bottom of a test tube.

TABLE No. 9.—*Milk from healthy cow (No. 2) inoculated one and one-half hours after milking.*

[Organisms from 24 hour agar cultures. Controls, same milk heated to boiling.]

	Colonies per loop at once after inoculation.	Colonies per loop after 8 hours at 37° C. moderate shaking.	Colonies per loop after 8 hours at 37° C. after vigorous agitation and mixing with pipette.
B. typhosus in raw milk	5,620	2,640	9,720
B. typhosus, control	9,540	[a] [b] 1,000,000	....
Staphylococcus pyogenes aureus in raw milk	1,660	3,810	5,610
Staphylococcus pyogenes aureus, control	1,850	[a] [b] 200,000	....
B. coli in raw milk	2,600	9,720	[a] [b] 100,000
B. coli, control	8,100	[a] [b] 1,500,000	....
Original milk	17	15	....

[a] Innumerable. [b] About.

TABLE No. 10.—*Milk from healthy cow (No. 2) inoculated one and one half hours after milking.*

[Controls, same milk heated in Arnold sterilizer to 100° C. for ten minutes. Organisms used were from 24-hour agar cultures.]

	Bacteria per loop at once.	Bacteria per loop after 8 hours at 37° C., tube moderately shaken.	Bacteria per loop after 8 hours at 37° C., culture vigorously agitated 5 minutes and mixed with a pipette.
B. lactis aerogenes in raw milk	1,200	4,100	14,000
B. lactis aerogenes, control	1,200	([a])	....
B. typhosus in raw milk	3,440	4,107	7,560
B typhosus, control	2,160	([a])	....
Staphylococcus pyogenes aureus in raw milk	2,120	1,220	2,070
Staphylococcus pyogenes aureus, control	2,660	30,000	....
Original milk	4	6	....

[a] Innumerable

The next experiment was designed to show whether the phenomenon of germicidal action could not be duplicated by the addition of typhoid agglutinating serum to milk that had been boiled.

TABLE No. 11.—*Milk from healthy cow (No. 2) planted one and one-half hours after milking.*

[Controls, same milk boiled—24-hours old agar culture used.]

	Bacteria per loop at once after inoculation.	Bacteria per loop after 2 hours at 37° C.	Bacteria per loop after 4½ hours at 37° C.	Bacteria per loop after 6½ hours at 37° C.	Bacteria per loop after 8½ hours at 37° C.	
					Moderately shaken.	Mixed with pipette.
B. typhosus in raw milk	1,880	1,380	1,060	1,480	1,980	12,200
B. typhosus in same milk boiled (control)	2,120	1,620	[a] [b] 800,000	([a])	([a])	([a])
B. typhosus in raw milk plus 1 drop typhoid serum	2,100	2,040	1,920	2,360	1,260	[a] [b] 20,000
B. typhosus in boiled milk plus 1 drop typhoid serum	2,280	2,360	7,020	6,480	10,860	[a] [b] 66,000
B. typhosus in bouillon plus 1 drop typhoid serum	1,830	970	2,920	9,180	11,160	[a] [b] 100,000
Original milk	0	1	2	2	2	16

[a] Innumerable. [b] About.

The typhoid serum used in this experiment was a strongly agglutinating horse serum (strength over 1 : 10,000).

It will be seen that boiled milk plus typhoid agglutinin acts much the same as the raw milk. The contrast in each case with the control (boiled milk) is striking.

The fact that agglutination plays a prominent rôle in this phenomenon is well illustrated in this table by the fact that the bacterial clumps may be shaken apart. This was confirmed by the microscopic examinations of stained smears in each case.

TABLE No. 12.—*Milk from healthy cow (No. 2).*

[Plantings one hour after milking.]

	Bacteria per loop at once after inoculation.	Bacteria per loop after 3 hours at 37° C.	Bacteria per loop after 5 hours at 37° C.	Bacteria per loop after 7 hours at 37° C.	
				Moderately shaken.	Mixed with pipette.
B. typhosus in raw milk	5,400	4,680	4,720	2,250	5,400
B. typhosus in boiled milk	6,400	5,040	[a] [b] 100,000	[a] [b] 200,000	[a] [b] 200,000
48-hour B. coli culture in raw milk	16,000	12,000	11,000	9,000	[a] [b] 40,000
Original milk	2	1	2	3	112

[a] Innumerable.

[b] About

TABLE No. 13.—*Milk from healthy cow (No. 2).*

[Plantings one hour after milking.]

	Bacteria per loop at once after inoculation.	Bacteria per loop after 3 hours at 37° C.	Bacteria per loop after 5 hours at 37° C.	Bacteria per loop after 7 hours at 37° C.	
				Moderately shaken.	Mixed with pipette.
1 loop fæces and hay emulsion in raw milk.	720	660	480	420	4,320
1 drop fæces and hay emulsion in raw milk.	4,860	4,640	6,000	3,780	9,720
[illegible] drops fæces and hay emulsion in raw milk	10,500	7,500	9,900	13,000	20,000

TABLE No. 14.—*Milk from healthy cow (No. 2) planted one and one-half hours after milking.*

[Controls same milk boiled.]

	Bacteria per loop at once after inoculation.	Bacteria per loop after 2½ hours at 37° C.	Bacteria per loop after 4½ hours at 37° C.	Bacteria per loop after 6½ hours at 37° C.	Bacteria per loop after 8½ hours at 37° C.	
					Moderately shaken.	Mixed with pipette.
1 drop cow fæces and hay suspension in raw milk	0	0	3	26	14	180
1 drop cow fæces and hay suspension in boiled milk (control)	11	13	18	80	1,260	...
4 drops cow fæces and hay suspension in raw milk	51	[illegible]	124	150	1,800	5,400
4 drops cow fæces and hay suspension in boiled milk (control)	7	[illegible]	48	720	3,300	......
Original milk	0	1	2	2	2	46

The fact that bacterial clusters may be separated by shaking, etc., is still more convincingly demonstrated in many of the other tables throughout the remainder of this article.

THE GERMICIDAL ACTION COMPARED WITH THAT OF BLOOD SERUM.

For the purpose of comparison the following experiment was made with fresh blood serum. The blood was drawn from the jugular vein of a horse, defibrinated and centrifugated for fifteen minutes at 1,800 revolutions per minute. In this way a fresh serum free from fibrin and cellular elements was quickly obtained. Care was exercised throughout the process to avoid contamination.

The serum was now divided into two portions: (1) Untreated, and (2) heated to 60° C. for twenty minutes. This temperature was selected as being sufficient to destroy the bacteriolytic property without seriously interfering with the agglutinins.

The heated and the unheated serum was now inoculated with 24-hour-old cultures from agar slants. The bacillary emulsion was first drawn in and out of a pipette in order to break up clumps.

	Bacteria per loop—					
	At once after inoculation.	After 2 hours at 37° C.	After 4 hours at 37° C.	6 hours at 37° C.		After 24 hours at 37° C.
				Moderately shaken.	Mixed with a pipette.	
B. typhosus in fresh horse serum	3,240	328	364	220	636	11,000
B. typhosus in fresh horse serum, heated to 60° C. 20 minutes	2,700	2,650	7,600	[a] [b] 70,000	[a] [b] 250,000	(a)
B. lactis aerogenes in fresh horse serum	1,500	5	0	0	0	5,400
B. lactis aerogenes in fresh horse serum, heated to 60° C. 20 minutes	2,640	3,180	9,000	[a] [b] 100,000	[a] [b] 200,000	a

[a] Innumerable. [b] About.

It is at once evident that there is a general resemblance between blood serum and milk so far as this phenomenon is concerned. It is also plain that blood has a much quicker and stronger action than milk.

The results of the bacterial counts upon agar plates were confirmed by microscopical examination of stained smear preparations. At first the organisms were well distributed throughout the serum, whether heated or unheated. There were no clumps of over six or eight individuals.

At the end of six hours no organisms could be found under the miscroscope in preparations made from the unheated serum planted with *B. lactis aerogenes*. Only occasionally could the typhoid bacil lus be discovered in the corresponding serum at the end of six hours. This agrees with the number of colonies upon the agar plates.

The heated serums gave quite a different picture under the microscope. Many organisms were found, lying singly, in small and long chains, and in dense clusters. This corresponded to the innumerable growth upon the agar plates.

RELATION TO PHAGOCYTOSIS.

Milk contains many leucocytes and it therefore seems reasonable to assume that active phagocytosis takes place in the fresh raw product. A priori it seemed to us that this might account for the germicidal property of milk. This assumption was apparently confirmed when we found that stained smear preparations showed but few if any bacteria in the cells in the fresh milk just after inoculation with bacterial cultures, while similar preparations made from the same milk eight hours later, kept at 37° C., showed numerous bacteria in some of the cells.

The following experiments, however, demonstrate that the germicidal power of milk is independent of its cellular contents. The leucocyte-free milk is quite as active as the leucocyte-rich sediment obtained by centrifugation.

Table No 15.

[Milk from a healthy cow (No. 2) was centrifuged for twenty minutes at 1,500 revolutions per minute. Part of the supernatant fluid was passed through a Berkefeld filter and a clear bluish serum obtained. Five sets of tubes were inoculated with 24-hour agar cultures, (1) the filtered clear serum, (2) the supernatant fluid free from leucocytes, (3) the sediment rich in leucocytes, (4) the original whole milk, and (5) sterilized milk. The inoculations were made three hours after milking.]

	Bacteria per loop, at once after inoculation.	Bacteria per loop after 8 hours at 37° C.	
		Shaken moderately before planting.	Vigorous agitation before planting.
B. typhosus in filtered milk serum	22,000	10,000	
B. typhosus in leucocyte-free supernatant fluid	19,000	1,900	
B. typhosus in leucocyte-rich sediment	32,000	3,600	
B. typhosus in whole raw milk	3,500	1,200	15,000
B. typhosus in sterilized milk (control)	16,000	[a] [b] 160,000	
B. lactis aerogenes in filtered milk serum	33,000	[a] [b] 330,000	
B. lactis aerogenes in leucocyte-free supernatant fluid	4,000	210	
B. lactis aerogenes in leucocyte-rich sediment	42,000	740	
B. lactis aerogenes in whole raw milk	5,400	33	92
B. lactis aerogenes in sterilized milk (control)	36,000	[a] [b] 360,000	
Original milk	19	42	

[a] Innumerable.

[b] About.

This table also shows the effect of shaking the milk in breaking up bacterial clusters.

TABLE No. 16.

[The milk was brought to the laboratory and inoculated with the cultures two hours after milking. The time given, however, is from the hour of inoculation. When obtained, the whole milk contained 1,200 bacteria per cubic centimeter. The sediment, rich in leucocytes, contained 630 bacteria per cubic centimeter, and the milk serum 490. The milk was revolved in a centrifuge at 1,500 revolutions for twenty minutes; the sediment pipetted off was rich in leucocytes. The supernatant serum was drawn off and filtered three times through asbestos and was therefore free of leucocytes. The milk was inoculated with a pure culture of *B. lactis aerogenes* and *B. typhosus*. Four test tubes were prepared with each culture, (1) whole milk, (2) the sediment rich in leucoytes, (3) the milk serum containing no leucocytes, and (4) sterilized whole milk used as a control.]

	Bacteria per loop, at once after inoculation.	Bacteria per loop after—		
		3 hours at 37° C.	6 hours at 37° C.	9 hours at 37° C.
B. lactis aerogenes:				
Whole milk	1,500	30	2	46
Sediment, rich in leucocytes	1,200	31	5	34
Milk serum, no leucocytes	1,400	52	8	370
Control, sterilized milk	1,300	25,000	200,000	(a)
B. typhosus:				
Whole milk	7,200	5,000	1,500	1,700
Sediment, rich in leucocytes	6,100	2,400	2,300	1,600
Milk serum, no leucocytes	4,200	2,800	3,900	14,000
Control, sterilized milk	3,700	15,000	67,000	(a)

[a] Innumerable.

The milk in both of these experiments was obtained from a healthy cow (No. 2) in a cleanly manner, but without special precautions.

The tables give the number of bacteria per loop on agar, grown at 37° C. and counted after twenty-four hours.

These tables eliminate the leucocytes and phagocytosis as a material factor in the problem we are studying.

IS THE GERMICIDAL ACTION SPECIFIC?

Almost all those who have studied this part of the problem conclude that the germicidal action of milk is specific. The following experiment confirms these observations. Here we have the same milk showing an active power of restraining the growth of typhoid and staphylococcus pyogenes aureus, but not paratyphoid A or B.

Reference to our work upon the influence of heat (*vide infra*) upon this property of milk also indicates its specific nature.

The table again demonstrates that agglutination plays at least some part in the apparent decrease.

Table No. 17. *Milk from healthy cow (No. 2).*

[Inoculated one and one half hours after milking. Controls, same milk heated to boiling. Organisms from 24 hour agar culture, using suspension in water of condensation in test tube.]

	Bacteria per loop at once after inoculation	Bacteria per loop after 10 hours at 37° C.	
		Test tube moderately shaken.	Vigorous agitation of culture for 5 minutes.
B. typhosus in raw milk	1,760	1,700	12,500
B. typhosus in control	2,100	(a)	....
B. para typhosus A in raw milk	2,740	(a)	(a)
B. para typhosus A in control	2,300	(a)	....
B. para typhosus B in raw milk	2,460	(a)	(a)
B. para typhosus B in control	3,200	(a)	....
Staph. pyog. aureus in raw milk	1,420	900	9,100
Staph. pyog. aureus in control	1,830	6,640	....
Original milk	7	42	....

[a] Innumerable.

THE EFFECT OF DILUTION.

Whole raw milk from a healthy cow (No. 2) was diluted in the proportion of 1 volume of milk to 3 volumes of distilled water. Inoculations were made two hours after milking; 24-hour agar cultures used.

Table No. 18.

	Bacteria per loop at once after inoculation.	Bacteria per loop after 10 hours at 37° C.	
		Tube shaken moderately.	Culture vigorously agitated 5 minutes and mixed with a pipette.
B. typhosus in diluted milk (1+3)	11,000	3,000	25,000
B. typhosus in same milk undiluted	35,000	1,200	15,000
B. typhosus in sterilized milk (control)	16,000	[a, b] 160,000	....
B. lactis aerogenes in diluted milk (1+3)	3,000	1,000	67,000
B. lactis aerogenes in same milk undiluted	5,400	33	92
B. lactis aerogenes in sterilized milk (control)	36,000	[a, b] 360,000	....

[a] Innumerable.

[b] About.

TABLE No. 19.—*Milk from a healthy cow (No. 2), inoculated one and one-half hours after milking.*

[Controls, same milk heated to 100° C. in Arnold sterilizer for ten minutes. Organisms from 24-hour agar cultures, suspended in normal salt solution.]

	Bacteria per loop at once after milking.	Bacteria per loop after 7½ hours at 37° C. Tube shaken moderately.
B. lactis aerogenes in raw milk diluted with same milk boiled (raw 3 parts, boiled 1 part)	1,460	9,000
B. lactis aerogenes in same milk undiluted	6,000	30,000
B. lactis aerogenes (control, i. e., boiled milk)	1,200	(*a*)
B. typhosus in raw milk diluted with boiled milk as above (3+1)	2,040	2,100
B. typhosus in same milk undiluted	1,680	2,340
B. typhosus (control, i. e., boiled milk)	2,160	*a*
Staphylococcus pyogenes aureus in raw milk diluted with boiled milk as above (3+1)	1,060	1,530
Staphylococcus pyogenes aureus in same milk undiluted	1,290	1,320
Staphylococcus pyogenes aureus (control, i. e., boiled milk)	2,660	30,000
Original milk	3	430

a Innumerable.

Dilution has an appreciable effect. For instance, in Table No. 18 there was a reduction of 96.6 per cent in the number of colonies of typhoid in whole milk, but only 73 per cent in the diluted milk. A similar effect is shown in relation to *B. lactis aerogenes.*

These experiments were originally designed to demonstrate the presence of a germicidal substance in solution, but the results again show that the action is in part at least due to agglutination. As far as the restraining action is concerned, the results resemble a feeble antiseptic rather than a germicide.

THE EFFECT OF HEATING AND FREEZING.

The effect of heat varies with the micro-organism. The power to restrain *B. lactis aerogenes* is weakened and destroyed before that for typhoid. Thus, 55° C. for thirty minutes or 60° C. for twenty minutes markedly alters or destroys the power of milk to restrain the *B. lactis aerogenes*, while this degree of heat shows little influence as far as *B. typhosus* is concerned.

Freezing milk for ten minutes apparently does not influence its germicidal power. Freezing the milk for forty-eight hours before inoculating it has no influence upon its restraining action as far as the typhoid organism is concerned, but apparently destroys this power for *B. lactis aerogenes.*

In the freezing experiments a small quantity of the milk was frozen solid by a mixture of salt and ice and kept in this condition for the periods stated. It was then cautiously thawed and inoculated with the micro-organisms to be tested.

The reason for selecting the temperatures used in Table No. 20, viz, 55° C. and 65° C., was to differentiate the true bactericidal from the agglutinating substances, based upon similar work upon blood serum.

TABLE No. 20.—*Milk from a healthy cow (No. 2) inoculated two hours after milking.*

	Bacteria per loop at once after inoculations.	Bacteria per loop after 8 hours at 37° C.
B. typhosus in milk first frozen 10 minutes	([a])	800
B. typhosus in milk heated to 60° C. 20 minutes	8,000	12,000
B. typhosus in milk heated to 80° C. 20 minutes	18,000	[a] [b] 380,000
B. typhosus in the whole raw milk	3,500	1,200
B. typhosus in sterilized milk (control)	16,000	[a] [b] 160,000
B. lactis aerogenes in milk first frozen 10 minutes	10,000	1,700
B. lactis aerogenes in milk heated to 60° C. 20 minutes	11,000	[a] [b] 50,000
B. lactis aerogenes in milk heated to 80° C. 20 minutes	20,000	[a] [b] 400,000
B. lactis aerogenes in the whole raw milk	5,400	33
B. lactis aerogenes in sterilized milk (control)	36,000	[a] [b] 500,000
Original milk	19	42

[a] Innumerable.

[b] About.

TABLE No. 21.—*Milk from healthy cow (No. 2) inoculated two hours after milking.*

[24 hour culture used in each case.]

	Colonies per loop at once after inoculation.	Colonies per loop after 10 hours at 37° C.	
		Shaken	Vigorously agitated.
B. typhosus in raw milk untreated	1,070	1,960	4,300
B. typhosus in milk heated to 55° C. 30 minutes	1,350	2,400	17,820
B. typhosus in milk heated to 65° C. 30 minutes	960	13,000	
B. typhosus in milk boiled 1 minute	1,150	[a]	
B. lactis aerogenes in raw milk untreated	820	12,000	15,000
B. lactis aerogenes in milk heated to 55° C. 30 minutes	1,400	[a]	
B. lactis aerogenes in milk heated to 65° C. 30 minutes	400	([a])	
B. lactis aerogenes in milk boiled 1 minute	950	[a]	
Original milk	2	18	

[a] Innumerable.

[b] About.

ABLE No. 22.—*Milk from healthy cow (No. 2) inoculated three hours after milking.*

[24-hour agar culture used in each case.]

	Bacteria per loop at once after inoculation.	Bacteria per loop after 9 hours at 37° C.	
		Shaken.	Vigorously agitated.
. typhosus in whole raw milk	6,500	2,300	14,000
. typhosus in whole milk heated to 60° C. 20 minutes	10,000	3,400	
. typhosus in sterilized milk (control)	9,000	[a][b] 200,000	
. lactis aerogenes in whole raw milk	5,300	325	1,700
. lactis aerogenes in milk heated to 60° C. 20 minutes	7,000	11,000	
lactis aerogenes in sterilized milk (control)	7,500	[a][b] 150,000	
riginal milk	18	73	

[a] Innumerable. [b] About.

TABLE No. 23.—*Milk from healthy cow (No. 2) inoculated one and one-half hours after milking.*

[24-hour agar culture in each case.]

	Bacteria per loop at once after inoculation.	Colonies per loop after 10 hours at 37° C.	
		Shaken.	Vigorously agitated.
B. typhosus in whole raw milk	1,000	3,500	19,500
B. typhosus in whole milk heated to 60° C. 30 minutes	1,700	18,000	43,000
B. typhosus in whole milk heated to 70° C. 30 minutes	2,100	[a][b] 200,000	[a][b] 300,000
B. typhosus in whole milk first frozen for 48 hours	1,520	1,020	
B. typhosus in milk boiled for 1 minute (control)	1,800	[a][b] 400,000	
B. lactis aerogenes in whole raw milk	2,000	29,000	75,000
B. lactis aerogenes in whole milk heated to 60° C. 30 minutes	1,670	[a]×100	[a]×100
B. lactis aerogenes in whole milk heated to 70° C. 30 minutes	870	[a]×100	[a]×200
B. lactis aerogenes in whole milk first frozen 48 hours	1,040	[a]× 20	
B. lactis aerogenes in whole milk boiled for 1 minute (control)	3,000	[a]× 30	
Original milk	13	210	

[a] Innumerable [b] About.

× Means about the stated number of times the number of colonies shown in the first column.

REVIEW OF THE LITERATURE UPON THE SUBJECT.

It will be seen from the following review of the literature upon this subject that our work confirms the facts which have been recorded by some others. There is, however, a disagreement concerning the interpretation of these facts. One class of observers, while admitting that there is a primary reduction of bacteria in fresh raw milk, believes this to be entirely independent of any germicidal action of that fluid. The reduction is attributed to other causes, such as a "restraining" action of the milk, inhibition resulting from strange media, etc. Others believe that fresh raw milk possesses definite, though feeble, germicidal properties. Some of the authorities cited absolutely deny not only the germicidal but also the restraining power of milk, claiming a steady increase in numbers from the start.

Fokker[a] was the first to call attention to the bactericidal properties of milk. He was led to investigate this subject through the results of Nuttall, Buchner, and Lubarsch, who found that blood contained substances capable of destroying bacteria. Fokker obtained goats' milk under careful aseptic precautions and divided it into two portions, one of which was heated, and both portions then infected with bacteria of souring milk. The cooked portion would always sour within twenty-four hours, while the fresh, unheated portion would keep at least, and sometimes more than, three or four days.

He also noted that by the use of cultures on plates there was first a diminution in the number of bacteria in milk. He further found that the brief heating of milk did not always destroy its bactericidal properties, but that prolonged heating at 70° C. is sufficient to destroy this action.

Uffelmann[b] observed the multiplication of cholera vibrios during the first twelve to sixteen hours in raw milk to be less than in the controls with cooked milk.

Weigmann and Zirn[c] also investigated the question of cholera vibrios in raw milk and found a diminution in the first four hours, but not in cooked milk. They believe that the presence of acid causes the killing of the vibrios.

A few years later, 1894, Hesse[d] confirmed the pioneer work of Fokker and stated most positively that cholera and typhoid are both killed by fresh milk and concludes from his studies as follows:

> The killing of the organisms begins the moment the cholera bacilli are added to the milk. This is complete, almost without exception, at room temperature (15°-20° C.) within twelve hours; at incubator temperature within six to eight hours. The destruction of the organisms is independent of the acid content of the milk, and independent of the milk organisms and their metabolic products. It is more probably to be looked upon as a vital function of the living milk, which is immediately lost upon heating to 100° C.

He, therefore, recommended the use of raw milk as a therapeutic measure in cholera.

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[a] Fokker, A. P.: Ueber die bacterienvernichtenden Eigenschaften der Milch. Fortschr. der Med., vol. 8, 1890, p. 7.

[b] Uffelmann, J.: Beiträge zur Biologie des Cholerabacillus. Berl. klin. Woch., vol. 29, 1892, p. 1209.

[c] Weigmann, H., and Zirn, Gg.: Ueber das Verhalten der Cholerabakterien in Milch und Molkereiprodukten.

[d] Hesse, Walter: Ueber die Beziehungen zwischen Kuhmilch und Cholerabacillen. Zeit. f. Hyg., vol. 17, 1894, p. 238.

Basenau,[a] 1895, disagrees with Hesse in that he could not confirm the destruction of cholera vibrios in raw milk, but found, on the contrary, that these organisms multiply vigorously from the start. He also experimented with *B. morbificans bovis* (enteritidis group) with the same result. He drew his milk from a healthy cow under special antiseptic precautions, but could not demonstrate definite bactericidal substances in the milk. At most Basenau believes that there is only a temporary restraining power, and he found that a similar restraining action was present when bacteria were transplanted into nutrient bouillon, and therefore this property is not specific for milk.

Heim[b] agrees with this view and adds to the list of organisms which are not affected typhoid and tubercle bacilli.

Schottelius,[c] Kitasato[d] and Friedrich[e] confirm the work of Basenau and Heim upon the lack of bactericidal properties of fresh milk.

Cozzolino[f] studied asses', cows', goats', and human milk, finding a reduction of organisms in a portion of the experiments, asses' milk being the strongest and goats' milk the weakest in bactericidal activity. Human milk is unique in its behavior toward *B. coli communis*, reducing the numbers materially during the first fourteen to twenty-four hours. Cozzolino, however, used milk which he endeavored to render sterile by heating to 55° to 58° C. for one hour on each of eight successive days.

Schenk[g] found a bactericidal substance in human milk, though in small quantities.

Hunziker,[h] 1901, showed that the action of the germicidal substance or condition varied with the individual cow and that its duration was influenced by the degree of temperature at which the milk was kept. The germicidal action was most rapid at comparatively high temperatures and the minimum number of bacteria was reached in a comparatively short time, while at the lower temperatures the intensity of the action was lessened and its duration was increased so that the minimum number was reached at a later period.

Klimmer,[i] as a result of his work, concludes that human milk lowers the number of organisms greatly, but that asses' milk develop-a specific bactericidal activity. He does not look upon the reduced number of bacteria as a result of the bactericidal activity, but believes it to be due rather to a change in the media.

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[a] Basenau, Fritz: Ueber die Ausscheidung von Bacterien durch die thätige Milch drüse und über die sogen. bactericiden Eigenschaften der Milch. Arch. f. Hyg., vol. 23, 1894, p. 14.

[b] Heim: Arb. Kais. Gesundhamte., vol. 4, p. 294.

[c] Schottelius: Centralbt. f. Bakteriol., v. 20, no 25, Dec., 1896, p. 897

[d] Kitasato, S.: Das Verhalten der Cholerabacterien in der Milch. Zeit. f. Hyg., vol. 5, 1889, p. 491.

[e] Friedrich: Arb. a. d. Kais. Gesndhtsamte., vol. 13, p. 465.

[f] Cozzolino: Arch. f. Kinderheilkunde, vol. 33, p. 405

[g] Schenk: Monattsschr. f. Geb. u. Gyn., vol. 19, 1904.

[h] Hunziker: Cornell Univ. Agr. Exper. Sta., Bull. 197, 1901.

[i] Klimmer: Arch. f. Kinderheilkunde, vol. 36, 1903, p. 1.

Moro[a] denies all bactericidal properties of raw milk so far as the cholera, typhoid, and colon bacteria are concerned. This he found true of cow's as well as of woman's milk. He found, however, that the serum of breast-fed children had greater bactericidal properties than the serum of children raised on cooked milk.

Park[b] studied this question in 1901 and concluded that freshly drawn milk contains a slight and variable amount of chemical substances which are capable of inhibiting bacterial growth. At temperatures under 50° F. these substances act efficiently, unless the milk is filthy, for from twelve to twenty-four hours, but at higher temperatures their effect is very soon completely exhausted, and the bacteria in such milk will then rapidly increase. Thus the bacteria in fresh milk which originally numbered 5,000 per cubic centimeter decreased to 2,400 in the portion kept at 42° F. for twenty-four hours, but rose to 7,000 in that kept at 50° F., to 280,000 in that kept at 65° F., and to 12,500,000,000 in the portion kept at 95° F.

Park,[c] 1901, believes this property is too elusive to be of practical use in dairying.

Conn,[d] 1903, confirmed the fact that during the first six hours there is a diminution in the number of bacteria in raw milk, but leaves the question open whether we are dealing with germicidal properties in raw milk or whether the organisms are simply becoming more accustomed to their new medium.

Heinemann[e] reported some investigations upon the subject which may reconcile the results of the different writers. He finds that for certain species of bacteria there is a bactericidal substance in raw milk while for other species there is none. Moreover, this germicidal property does not assert itself after the milk is from five to seven hours old. This power is also destroyed after heating milk to 56° C. for thirty minutes or by bringing it to the boiling point.

Stocking,[f] 1904, investigated this question by studying the multiplication of certain groups of bacteria, and concluded that many of the species gaining access to the milk find the condition so different to their natural habitat that they are not able to multiply and therefore they drop out very soon. On the other hand, common lactic-acid organisms multiply more or less rapidly and continuously from the start. He believes that the reduction in the number of bacteria during the first few hours is not the result of any germicidal condition or property possessed by the milk, but simply the natural dropping out of those species which do not find the milk a suitable medium in which to develop.

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[a] Moro: Munch. med. Woch., vol. 48, Oct., 1901, p. 1770; also Arch. f. Kinderheilkunde, vol. 33, p. 435, 1902.

[b] Park, William H.: The great bacterial contamination of the milk of cities, can it be lessened by the action of health authorities? N. Y. Univ. Bull. of Med. Sci., vol. 1, 1901, p. 71.

[c] Park, William H.: N. Y. Univ. Bull. Med. Sci., vol. 1, 1901.

[d] Conn, W. H.: Bacteria in milk and its products. London, 1903, p. 98.

[e] Heinemann, Paul Gustav: The kinds of bacteria concerned in the souring of milk. Chicago, 1903.

[f] Stocking, W. A., Jr.: The so-called "germicidal property" of milk. Storrs Agric. Sta. Bull. 28, 1904, p. 89.

Behring,[a] 1904, in his recent publications claims that milk has similar bactericidal property to that possessed by the blood. Further, that these bactericidal substances are rendered inactive at 60° C. for one hour or 50° C. in vacuo. He believes that heating milk to 60° C. for sixty minutes appreciably weakens the immune bodies contained in it, and that the great mortality of infants in large cities has a direct relation to the use of cooked milk. He believes that the important point in infant feeding is to use milk in which the native antibodies are intact. He uses this as one of his arguments in advocating the use of formaldehyde to preserve milk.

Friedel, Kutscher, and Meinicke,[b] 1904, working under Kolle's direction, in Koch's Institute for Infectious Diseases, at Berlin, found as a result of numerous experiments that fresh raw milk contains bactericidal properties, similar to those of fresh blood serum, against the cholera vibrio. But no such property was found as far as the typhoid, paratyphoid, and dysentery bacilli, the organism of meat-poisoning, and *B. coli* are concerned.

They found that fresh raw milk has a feeble property of restraining the growth of the dysentery bacillus. This property is not destroyed by heating the milk to 60° C. for one hour, but is destroyed above 70° C. These investigators believed that this property of milk in question is a restraining action and not a bactericidal one, especially in view of their dilution experiments.

They found that the bactericidal property of milk, as far as the cholera organism is concerned, is weakened by heating the milk to 60° C. and by the addition of hydrochloric acid, pepsin, and sputum.

Knox and Schorer[c] find that neither raw nor pasteurized milk seems to exert any definite deterrent action upon the growth of the dysentery bacillus, and conclude that it is evident that the much talked of bactericidal action of milk is of little or no aid in maintaining the low count desired in a milk used in infant feeding.

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[a] Behring, E.: Säuglingsmilch und Säuglingsterblichkeit. Therapie die Gegenwart, n. s., vol. 4, 1904, p. 1-10.

[b] Untersuchungen über die bakteriziden und entwickelungshemmenden Wirkungen der rohen und der auf verschiedene Temperaturen erwarmten Milch gegen über den oben gennanten Bakterien. Klinische Jahrbuch, vol. 13, 1904-5, p. 328.

[c] Knox, J. H. Mason, and Schorer, Edwin H.: A study of hospital and dispensary milk in warm weather, with special reference to pasteurization. Arch. of Pediatrics, July, 1907.

[Since publishing our article the following additional references to the literature have come to our notice:]

Coplans[a] points out that two phenomena must be considered when organisms are transferred to a new medium. First, a period of latency due to change of environment (nature of food supply, reaction, and temperature). During this period of latency there is little or no increase in number. The second phenomenon is one observed in the case of fresh raw milk. It is a bactericidal inhibitory property.

He showed that during the first six hours there is a reduction in the number of *B. coli* in raw milk, but no change in the number in boiled milk. At the end of twenty-four hours the increase in numbers in boiled milk is twenty times as great as in raw milk; at the end of forty-eight hours there is little difference in the numbers in raw and boiled milk. He holds that a given quantity of fluid can harbor only a certain number of organisms; therefore the number of bacteria planted has something to do with the rapidity of growth.

An interesting point demonstrated by Coplans is the fact that the addition of enough boric acid to merely check the growth of organisms in milk abolishes all bactericidal property of the fluid.

Moro,[b] in a recent investigation, found that unfiltered raw cow's milk always effected a diminution in the number of colonies of *B. typhosus* during the first few hours. He believes this action is due to a true bacteriolytic alexin. Human milk never brought about any actual reduction in the number of *B. typhosus*, but restrained the multiplication as compared with the boiled milk. He found that passing cow's milk through a Berkefeld filter deprived it of the power to reduce the number of bacteria or to restrain their multiplication.

St. John and Pennington[c] found that the heating of milk to 79° C. not only took away the power to bring about an initial decrease in the number of organisms, but destroyed the power to restrain the multiplication of bacteria throughout the period of observation, which was to the souring point. They call attention to the great care necessary in handling milk after commercial pasteurization, as when infected the growth of organisms is much greater than in milk not subjected to the process. The raw milk usually remained sweet twenty-four hours longer than pasteurized milk reinfected with organisms from the raw milk. They considered organisms only that were normally present in milk.

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[a] Coplans, Lancet, pp. 1074-1080. October 19, 1907.

[b] Moro, Zeit. f. exp. Path. u. Therap., pp. 470-479. Berlin, 1907.

[c] St. John and Pennington, Journal of Infectious Diseases, vol 4, No. 4, pp. 647-656. 1907.

Evans and Cope [a] have recently experimented upon the bactericidal property of milk and have reached the following conclusions:

1. Freshly drawn milk possesses a bactericidal activity toward certain micro-organisms and an inhibitory activity toward others.

2. This activity is destroyed at 68° C. and materially injured at 55° C. It varies in different cows and lasts from six to twelve hours.

3. Coagulation and acidity of milk do not depend solely upon the bacterial content. They are influenced by natural properties of milk, which are soon overshadowed by the metabolic products of bacteria.

4. Sterile cow's milk freshly drawn is acid to phenolphthalein and increases very slowly in acidity independent of bacterial metabolism, due probably to the destruction of colostrum cells.

5. Results obtained in testing milk with mixed bacterial floræ are influenced by bacterial antagonism.

Rullmann and Trommsdorff [b] state that they made a number of tests concerning the bactericidal properties of fresh milk. They state however, that unfortunately their experiments have not yet given them as clear a picture as they would wish of this apparently rather complicated phenomenon. However, they state that it seems justifiable to draw certain conclusions from their work without giving any of the details of the experiments. The conclusions follow:

(1) That the bactericidal power of milk is increased when it comes from udders the seat of mastitis (increased secretion of serum and also of alexin? influence of bacteria upon the (local?) production of immune bodies in the milk?).

(2) That there is a direct relation between the bactericidal property of milk and the number of leucocytes that it contains.

In a recent study upon the germicidal action of cow's milk Heinemann and Glenn [c] conclude as follows:

The decrease of bacteria in fresh cow's milk is more decided if fairly large numbers are inoculated than if small number only are present.

The relative increase of bacteria in milk is more pronounced in milk heated to 75° C. or 100° C. than in raw milk or milk heated to 56° C.

The difference in the relative decrease in numbers of bacteria in milk moderately shaken and vigorously shaken is not marked if this shaking is done by hand. Some difference was observed, however, and this difference might be more pronounced if the milk were shaken more violently.

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[a] The bactericidal property of milk, by Joseph S. Evans and Thomas A. Cope, University of Pennsylvania Medical Bulletin, Vol. XXI, pp. 264-274. 1908.

[b] Milchhygienische Untersuchungen, W. Rullmann and R. Trommsdorff, Archiv für Hygiene, Vol. LIX, pp. 221-265. 1906-7.

[c] Heinemann, P. G., and Glenn, T. H.: "Experiments on the germicidal action of cow's milk." Journ. Infec. Dis., Vol. V, pp. 534-541, Dec. 18, 1908.

Some species occurring naturally in milk decrease considerably in numbers during the first four or five hours, some decrease slightly, some hold their own or even increase.

Milk inoculated with pure cultures of bacteria seems to restrain to a marked degree the multiplication of these bacteria for several hours at 37° C. and for a somewhat longer period at room temperature, excepting in the case of *Strept. lacticus*, which increases from the beginning, although it may be inhibited to some extent.

Heating milk to 56° C. for thirty minutes does not entirely destroy the power to restrain the multiplication of bacteria: this power is weakened, however, and at 75° C. is destroyed entirely. This fact, together with the fact that milk serum agglutinates some species of bacteria *in vitro* to a marked degree, seems to favor the assumption that agglutinins are in part responsible for the apparent decrease of bacteria in fresh milk, since bactericidal substances are destroyed by heating to 56° C. for thirty minutes.

The agglutination of certain bacteria in milk serum seems to bear some relation to the apparent decrease in numbers of bacteria observed in fresh milk, but this is probably not the only factor causing such reduction.

SUMMARY AND CONCLUSIONS.

Judged by the number of colonies that develop upon agar plates, the bacteria in milk first diminish then increase in number. This so-called germicidal property of milk occurs only in the fresh raw fluid.

For the most part, our work plainly shows that no actual reduction in the number of bacteria occurs. However, when compared with the controls a restraining action is evident. The phenomenon therefore appears to resemble that of a weak antiseptic rather than that of a true germicide.

When milk is kept warm (37° C.), the decrease is pronounced within the first eight or ten hours. After this time the milk has entirely lost its restraining action.

When the milk is kept cool (15° C.), the decrease is less marked, but more prolonged.

The decrease in the number of bacteria is largely apparent, being due at least in part to agglutination.

The bacterial clusters may, to a certain extent, be shaken asunder. This fact goes far to reconcile the discordant results of the various investigations upon the germicidal properties of milk. Those who used dilution methods with vigorous agitation broke up the bacterial clusters, and thus obtained a larger number of colonies upon agar plates than those who plated directly with different technique.

Some of the leucocytes in milk seem to possess the power of phagocytosis, judged by microscopic preparations. Phagocytosis, however, plays no essential part in the "germicidal" action of milk, for the decrease in numbers is quite as marked in the cell-free milk as in the sediment rich in leucocytes.

The germicidal action of milk is specific. For instance, one sample restrained typhoid and *Staphylococcus pyogenes aureus*, but not paratyphoid A or B.

Dilution experiments demonstrate the enfeeblement of agglutinins rather than the presence of a germicidal substance in solution.

The germicidal actions of blood and milk resemble each other in some particulars. Blood serum acts more quickly and much more powerfully than milk.

Freezing milk for ten minutes and then thawing it does not affect the phenomenon in question. In one experiment freezing for forty-eight hours did not influence its restraining action upon typhoid, but destroyed it for *B. lactis ærogenes.*

Boiling milk or heating it above 80° C. destroys its "germicidal" properties. The effect of lesser degrees of heat varies with the micro-organism. Thus, the restraining action for *B. lactis ærogenes* is weakened by first heating the milk at 55° C. and almost destroyed at 60° C.; for typhoid it is not affected by heating the milk at 60° C. for twenty minutes, but is materially influenced at 70° C. for thirty minutes.

The "germicidal" action of milk varies in different animals and in the milk from the same animal at different times. At most, the action is variable and feeble. It can not take the place of cleanliness and ice, but may be taken advantage of in good dairy methods.

13. THE SIGNIFICANCE OF LEUCOCYTES AND STREPTOCOCCI IN MILK.

THE SIGNIFICANCE OF LEUCOCYTES AND STREPTOCOCCI IN MILK.

By William Whitfield Miller,[a]
Assistant Surgeon, Public Health and Marine-Hospital Service.

In the search for better and simpler methods for detecting milk from diseased cows, especially when mixed with the milk from healthy cows, as may be the case in market milk, particular attention has been paid to the presence of leucocytes and streptococci. In America, and to some extent abroad, much work has been done by State and city health authorities in the examination of dairy milk, with a view to determining the significance of these elements, and fixing standards limiting their number in acceptable dairy milk. It was early observed that in the milk of cows with udder disease of an inflammatory nature, pus cells and streptococci were almost invariably present in large numbers. Following this observation, examinations were made to determine how often leucocytes and streptococci were present in market milk. The result showed that they were present in the majority of milks to a greater or less extent. They were not so numerous, however, as in the milk of diseased animals. The interpretation placed upon these findings—viz, that one or more of the cows of the dairy herd from which the milk was derived was affected with garget (an inflammation of the udder)—is no longer regarded as strictly correct, since it is now well proven that the normal milk of healthy cows always contains leucocytes and usually streptococci. In the last two or three years work has been done that throws a new light on the subject and explains some of the discordant results previously obtained.

Whether the polymorpho-nuclear cells found in all milk shall be regarded as leucocytes or pus cells has been a subject of much dispute. As they were first looked upon as pus cells, it is readily understood why repugnance was felt at the idea of taking them in food, even if they were harmless. The earlier observers detected these cells in a large number of specimens of milk, but not in all, and concluded that they indicated some degree of inflammation of the udder. They fixed an arbitrary number (to a certain quantity of milk) as a limit, beyond which the milk was regarded as unfit for use. In other words, it was only a question to what extent the evil was to be endured. The present views concerning cellular elements in cows' milk are somewhat different.

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[a] This article is reprinted without revision, owing to Doctor Miller's death, November 24, 1908.

Histologists long ago pointed out that leucocytes occur in milk, when the gland structure showed no evidence of inflammation. Colostrum corpuscles are regarded as leucocytes loaded with débris of gland cells removed to clear up the channels for milk flow. It seems but natural that such a remarkable and rapid metabolic process as milk formation should be accompanied by a large number of leucocytes. In fact, in sections of normal functionating mammary glands they are found to be numerous in the capillaries and in the connective tissue spaces between the alveoli, from which they make their way between the gland cells and appear in the milk.

As regards leucocytes and pus cells there is no intrinsic difference. A leucocyte in fluids other than blood plasma—as milk, for example—soon undergoes changes which render it indistinguishable from a pus cell. That pus does occur in milk from inflamed mammary glands need scarcely be mentioned. Before considering further the significance of leucocytes, it will be of interest to note the various methods used for counting them. Stokes and Wegefarth[a] were the first to attempt an enumeration. Briefly, their method consisted in centrifuging a definite quantity of milk, spreading the entire sediment on a slide over a definite area, staining and examining with a $\frac{1}{12}$-inch lens; the number of leucocytes in ten or more fields of the microscope was noted, and an average struck. From this data an estimate was made of the number per cubic centimeter.

Stewart[b] and Slack[c] used a refinement of the same method. Doane-Buckley[d] modified the procedure in common use for counting blood cells, employing the Thoma-Zeiss instrument, with quite accurate results.

Savage[e] used a very similar method. Still another way of estimating the leucocytes is based on a procedure applied to blood—the use of a modified hematocrit, in which the milk is centrifuged and the leucocytes read on a scale, as sediment in volume per cent. Trommsdorff,[f] in Germany, has perfected this method. As might be expected different methods give widely different results. Bergey[g] in a recent paper records a number of comparative counts by the Stokes, Stewart,

[a] Medical News, 1897, No. 91, p. 45.

[b] American Medicine, 1905, No. 9, p. 486.

[c] Journ. of Infect. Diseases, 1906, Sup. No. 2.

[d] Maryland Agric. Exper. Station, 1905, Bull. 102.

[e] Brit. Med. Jour., 1905, No. 1, p. 1165.

[f] Munch. Med. Woch., 1906, No. 53, S. 541

[g] Univ. of Pa. Med. Bull., 1907, vol. 20, Sept.

Doane-Buckley, and Trommsdorf methods, which show great discrepancies. When making a number of counts from the same specimen of milk the Doane-Buckley technique gave the least variation and as compared with the others the highest counts. Russell and Hoffmann[a] in a similar comparison found the average variation 6 per cent using the volumetric, and 112 per cent using the smeared sediment technique. Most recent writers agree that the latter method should be abandoned as too inaccurate to be of service.

Concerning the number of leucocytes which have been found in the milk of healthy cows kept under the best conditions and counted by the most accurate methods, a great variation has been noted. Savage found numbers ranging from 35,000 to 4,500,000 per cubic centimeter, and more than three-fourths of the cows gave milk that averaged more than 100,000 per cubic centimeter. About the same figures were found for mixed milk from entire herds; more than four-fifths of the herds gave averages above 100,000 per cubic centimeter.

Doane found the number of leucocytes in the milk of a large number of cows to average more than 200,000 per cubic centimeter.

Russell and Hoffmann made a large number of counts from the milk of cows in which there was not the slightest clinical evidence of udder disease, nor had there been any history of such, and obtained results as high as 1,800,000, while 33 per cent gave counts higher than 500,000 per cubic centimeter; 83 per cent of those with slight udder disease or a history of garget gave counts of 500,000 per cubic centimeter or over.

Tests of milk from the same cow, taken from day to day, show great variations, although in general it may be said that a milk showing a high count one day will show a high count the next day, but not necessarily the next week or month; this, of course, when the health of the animal continues unchanged. The period of lactation seems to have no constant influence, except that the colostrum corpuscles are greatly increased after parturition. Bergey has shown that widely different counts are often found in the milk from different quadrants of the udder. Buchholtz,[b] Czerney,[c] and Michaelis[d] have pointed out certain facts relating to the secretion of milk that explain some of these discrepancies. They found that retention of milk in the gland ducts and alveoli for an unusual period causes a large increase in the number of leucocytes in the milk. When the milk is allowed to accumulate in the udder instead of being removed at regular and proper intervals, favorable conditions for the migration of leucocytes are found.

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[a] Journal of Infectious Diseases, 1907, Supplement No. 3, p. 63.

[b] Inaug. Diss., Gottingen, 1877.

[c] Prag. med. Woch., 1890, Bd. 15, S. 401-416.

[d] Arch. f. microsk. Anat., Bd. 51., S. 711.

It was deemed advisable by the early students of the subject, who considered all leucocytes in milk as "pus cells" and evidence of inflammation of the udder, to fix a limit to the number which might be allowed in a market milk. Stokes (loc. cit.) regarded an average of 5 cells to the field of the microscope, using his counting method, as indicative of pus. Bergey[a] adopted 10 cells per field as a standard. Stewart (loc. cit.) regarded 23 cells per field and Slack (loc. cit.) 50 cells as a proper standard, all using a modification of Stewart's technique. Doane advocates 500,000 leucocytes per cubic centimeter, Trommsdorff 10 volumes of sediment to 10,000 volumes of milk as a safe limit. Stewart's is the standard commonly employed in municipal health laboratories and corresponds roughly to 100,000 leucocytes per cubic centimeter. All of the above standards are arbitrary and are founded solely upon individual experience. The standard suggested by Trommsdorff limiting the amount of sediment in a centrifuged sample of milk seems valuable, not so much as an enumeration of the leucocytes as an indication of objectionable solid matter in suspension. It is of interest to note that although the number of leucocytes in milk from cows with diseased udders is usually much increased, Russell and Hoffmann have shown that this is not necessarily true, in some instances the count running below the average for normal milk. The daily variation in such cases often brings the count well within the usual normal limits. It seems likely that a numerical standard for leucocytes sufficiently high to include the milk of the greater majority of healthly cows would not be low enough to exclude in some cases the milk of cows with disease of the udder. Other and more definite signs of inflammation than that furnished by the leucocyte count alone must be sought.

Doane (loc. cit.) states that the occurrence of fibrin is positive proof of the existence of inflammation and has devised ways of demonstrating its presence in milk. The matter requires further study. The leucocytes per se can not be regarded as deleterious or foreign ingredients of milk; it is scarcely reasonable to expect that a food so distinctly animal in its origin should contain no organized elements.

The significance of pus in milk has been studied principally in connection with micro-organisms, looked upon as the exciting cause of the mastitis, with which the pus is associated. In the stained smears of milk sediment examined by the earlier investigators of leucocytes in cow's milk, attention was drawn to the large number of chain-forming micro-organisms (streptococci) present. They were especially numerous in milk that clearly contained pus or was known to be derived from cows with mastitis. At that time all streptococci were believed to be pathogenic, and it was assumed that when such organisms were found in milk they pointed to disease of the udder and were capable of propagating disease when ingested with the milk. Recent investigators have so clearly demonstrated the close relationship existing between the micro-organisms, which cause spontaneous souring of milk, and the streptococci that it will be of interest to consider the former at some length.

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[a] Bull. No. 125, Penna. Dept. Agric., 1904.

Pasteur, [a] Lister, [b] Hueppe, [c] and many others studied the souring of milk. Pasteur proved that micro-organisms were the cause, and Lister, twenty years later, isolated in pure culture a bacterium which he deemed the common agent. Subsequent bacteriologists have studied and described at length a great variety of bacteria found in ordinary dairy milk, capable of causing fermentation, with lactic acid formation. Many of these micro-organisms were later shown to have been introduced by unclean containing vessels and careless handling of the milk, and while capable of causing souring must be regarded as accidental and inconstant in occurrence. When drawn directly in sterile vessels with ordinary caution as regards cleanliness the variety of organisms is reduced to three or four. The technique of the early bacteriologist was somewhat imperfect, and cultural methods less exact than now. It is not surprising that identical bacteria were described by different observers as showing slight variations.

A review of the work done since Hueppe[d] in 1884 (who was the first to apply modern methods to the study of milk bacteria) shows that the common lactic bacteria may be classified in three groups. The first includes the bacilli of the type first described by Escherich,[e] the *Bacterium ærogenes* group. These organisms are classified with the colon group and owe their presence in milk to contamination with the feces of the cow.

Marpmann,[f] Grotenfeld,[g] Löffler,[h] Weigemann,[i] Kayser,[j] Clauss[k] and others have described organisms of this type. They grow readily on ordinary media with all the characteristics of the colon group, but do not as a rule produce indol. Although known since Hueppe's description as the *Bacterium acidi lactici* this organism is more commonly called *Bacterium ærogenes.*

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[a] Ann. d. Chim. et Phys., 3 Serie, 1858, 52.

[b] Quarterly Journal of Micros. Sc., 1878, 18, p. 177.

[c] Mitth. a. d. kaiserl. Gesundheitsamt, 1884, 2, 309.

[d] Mitth. a. d. kaiserl. Gesundh. amt., Bd. 2, S. 309.

[e] Darmbakterien des Sauglings, Stuttgart, 1886.

[f] Ergänzungshft. Centrbl. f. Allg. Gesundheitspfl., Bd. 11, S. 11.

[g] Fortschr. d. Mediz., Bd. IV, 1889.

[h] Berl. klin. Wochenschr., 1887, S. 631.

[i] Landw. Wochenbl. f. Schlesw. Holst., 1890, 29.

[j] Annal. de l'Institut Pasteur, 1894, p. 738.

[k] Inaug. Dissert., Würzburg, 1889.

The second group includes bacteria described by Clauss (loc. cit.), Günther and Thierfelder,[a] Esten,[b] Leichmann,[c] Kozai,[d] Schierbeck,[e] Haschmioto,[f] Harrison and Cumming,[g] Conn and Esten,[h] Hölling,[i] and Utz.[j] The characteristic organism is defined as oval or lance shape, occurring in pairs or short chains, colored by Gram, growing slowly on ordinary media, and producing no gas in presence of sugars. The growth on solid media is delicate and translucent. It causes rapid acid fermentation of milk, with coagulation. *Bacterium lactis acidi*, *Bacterium acidi lactici*, and *Bacterium Guntheri* are some of the terms used to designate this organism.

The third group includes micrococci described by Grotenfeld (loc. cit.) and Weigemann (loc. cit.) as streptococci. The cultural characters of this group are exactly similar to those of the second, the sole difference in the descriptions being in the morphology, in one case an oval or lance-shaped bacillus, in the other a streptococcus. Kruse[k] in 1903 pointed out the close similarity of organisms classed in the second and third groups and suggested that the difference of morphology was merely one of interpretation, namely, that the oval bacillus was a phase in the rapid growth by division of the streptococcus. Two years later, Heinemann,[l] after a careful comparison of strains of *Bacillus acidi lactici* from various sources with streptococci (sewage, pathogenic, water), concludef that they show no constant differences in growth, action on milk, or pathogenicity. Heinemann's work has done much to change existing ideas as to the significance of streptococci in milk, for he has shown that the most common organism of lactic acid fermentation, existing in practically all milk from healthy cows, is a streptococcus (*S. lacticus*, Kruse). Here again, as in the case of leucocytes in milk, it does not necessarily follow that streptococci when present are associated with disease. It does not seem strange that an organism so widely distributed in nature, o common on the skin and mucous membranes, and in the feces of nimals should be so often found in cows' milk.

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[a] Archiv. f. Hyg., Bd. XXV, S. 164, 1895.

[b] Ann. Report Storrs Agric. Exp. Station, 1896.

[c] Centralb. f. Bakt. 1896, Abt. II, Bd. 2, S. 799.

[d] Ztschr. f. Hyg., 1899, Bd. 31, S. 337.

[e] Arch. f. Hyg., 1900, Bd. 38, S. 294.

[f] Hyg. Rundschau, 1901.

[g] Jour. app. Microsc., 1902, Vol. 5, pp. 20–29.

[h] Ann. Report Storrs Agric. Exp. Station, 1902–3, p. 63.

[i] Inaug. Dissert., Bonn, 1904.

[j] Centralbl. f. Bakt., 1904, Abt. II, Bd. XI, S. 600.

[k] Centralbl. f. Bakt. Abt. I, Bd. 34, S. 737.

[l] Journ. of Infect. Diseases, 1906, Vol. 3, No. 2.

In their studies of leucocytes in smears made from milk, Conn and Esten (loc. cit.), Bergey (loc. cit.), Reed and Ward,[a] and others noted the number and frequency of occurrence of streptococci. As hey regarded the leucocytes as evidence of inflammation, they placed a similar construction upon the presence of these micro-organisms; since streptococci are well known to be a common cause of septic infection and pus formation. The recent investigations concerning the nature of the *Streptococcus lacticus*, a nonpathogenic organism, proving it to be indistinguishable, morphologically, from he pathogenic streptococci, show that such a conclusion is often incorrect. It is easily conceivable that in cases of garget and in septic conditions the streptococci associated with pus in the milk may be truly pathogenic. In fact, a number of writers in this country and abroad have described "outbreaks" following the use of milk containing streptococci from diseased cows.

Holst,[b] Stokes and Wegefarth,[c] Beck,[d] Lameris and Von Harrevelt,[e] Kenwood,[f] Savage,[g] and many others saw epidemics of sore throat with swelling of the cervical glands, colic, diarrhea, and fever lasting several days, which were ascribed to the use of milk from cows with garget. Such milk when examined was found to contain pus and streptococci in great abundance. Holst in an experiment upon himself drank 200 cubic centimeters of a culture of a streptococcus isolated from such a milk during an outbreak, and became ill with colic and diarrhea.

Petruschky and Kriebel[h] and Hölling[i] see in the streptococci found in milk a cause of summer diarrhea in children.

As regards the relationship between the numbers of leucocytes and streptococci, most writers agree that in mastitis the milk usually contains both in abundance. There is a difference of opinion concerning their relationship in milk from healthy cattle; Bergey and Trommsdorff finding a simultaneous increase or diminution, whereas Savage and others do not.

The manifest advantages to be gained from a knowledge of the pathogenic or nonpathogenic properties of the streptococci in milk has led to a number of investigations. Injections of milk or cultures isolated from milk into the tissues and peritoneal cavities of animals gave varying results. In some cases death of the animal ensued. It was found that as a rule when the organisms were pathogenic this property was gradually lost by cultivation on artificial media, and could be increased by carrying through a series of animals. Heinemann[a] in a recent study succeeded in raising the virulence of a number of strains of *Streptococcus lacticus*, by passage through successive rabbits, from almost nil to an equality with that of the *Streptococcus pyogenes*. Unfortunately the virulence for animals is not a certain index of the virulence for man.

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[a] Ref. Centralb. f. Bakt., Abt. 1, 1903, Bd. XXX, S. 83.

[b] Ref. Baumgartens Jahresber., 1895, S. 52.

[c] Med. News, 1897, vol. 71, No. 2, p. 45.

[d] Deutsches Vierteljahrschr. f. offentl. Gesundheitspfl. Heft III, S. 130.

[e] Zeitschr. f. Fleisch. u. Milch. Hyg., 1901, Bd. 11, S. 111

[f] Brit. Med. Journ. 1904, No. 1, p. 602.

[g] Journ. of Hyg., 1906, vol. 6, p. 123.

[h] Die Ursachen der Sommersterblichkeit der Säuglinge u. die Möglichkeit ihrer Verhütung. Leipzig, 1904.

[i] Inaug. Diss. Bonn, 1904.

Attempts to distinguish *S. lacticus* and *S. pyogenes* by hemolytic and agglutinative tests have been made by Schottmüller,[b] Lubenon,[c] Schlesinger,[d] and Müller[e] and Bergey (loc. cit.). No constant results have been obtained. Müller found that milk streptococci were almost as often hemolytic as the *S. pyogenes*, and heterologous strains were more strongly agglutinative than homologous. As has been pointed out, no specific characters have been revealed by cultivation on various media.

In view of the facts presented the assumption seems justified that the *Streptococcus pyogenes* and the *Streptococcus lacticus*, the common organism of lactic-acid fermentation, are indistinguishable by our present methods.

Briefly, the conclusions which present themselves are as follows:

(1) Many leucocytes and streptococci are present in the normal milk of a healthy cow.

(2) Leucocytes and streptococci are as a rule more numerous in the milk of diseased than in that of healthy cows.

(3) As an aid to veterinary inspection the number of leucocytes may furnish some information of value. If a dairy milk shows an unusually high leucocyte count, a special examination of the herd for garget, etc., should be made.

(4) No satisfactory method has been devised for distinguishing the pathogenic from the nonpathogenic streptococci in milk. Their significance is therefore a matter for further study.

(5) In view of the recent researches upon *Streptococcus lacticus* no constant relationship may be expected between the number of streptococci and the number of leucocytes in milk.

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[a] Journ. of Infect. Diseases, vol. 4, No. 1, 1907, p. 89.

[b] Münch. Med. Woch., 1903, Nr. 50, S. 909.

[c] Centralbl. f. Bakt., 1902.

[d] Zeitschr. f. Hyg., Bd. XLIV, 1903.

[e] Archiv f. Hyg., 1906, Bd. 56, S. 90.

14. CONDITIONS AND DISEASES OF THE COW INJURIOUSLY AFFECTING THE MILK.

CONDITIONS AND DISEASES OF THE COW INJURIOUSLY AFFECTING THE MILK.

By JOHN R. MOHLER, A. M., V. M. D.,
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IMPORTANCE OF A WHOLESOME MILK SUPPLY.

The reasons for securing a supply of pure and wholesome milk are so numerous and so important that the public should become acquainted with some of the more essential of them in order that assistance may be rendered in bringing about a satisfactory improvement. Public health demands the purity of all milk and milk products. Next to bread, milk is more extensively used as an article of diet than any other foodstuff. It forms a portion of the food of almost every person on practically every day of the year. Furthermore, unlike many other articles of diet, milk is consumed in most cases in an uncooked state, making it a very dangerous food should it perchance contain any deleterious organisms. Not only is milk a very suitable medium for almost every description of germ life which may gain access to it in its journey from the cow to the consumer, but it may also become contaminated while still in the udder through infectious or poisonous material present in the cow herself. In this paper, however, consideration will be given only to the latter aspect of the subject.

In this connection it will be necessary to keep in mind the requirements of an awakened public for a clean and wholesome milk, as well as the effect of any unreasonable or irrational demand upon the producer, which may cause him heavy losses or even to discontinue his business. It will also be apparent that in order to produce milk in compliance with the requirements hereafter described certain precautions must be taken, which will necessarily entail additional expense upon the producer of this higher grade of milk. The customer must therefore expect to pay his portion of any legitimate advance in the cost of production, and such increase in the price of milk due to its improved quality should be considered as money well expended.

Moreover, good milk of safe quality can not be had without a realization on the part of the farmer, the transportation agent, the dairyman, and the housewife of the danger in utilizing old, warm, or dirty milk. Education is therefore an important factor in the improvement of the milk supply, which can not be accomplished through laws and regulations alone. In view of these facts, it is recommended that the subject be taught in the schools, that popular articles be frequently prepared for the press, that lectures and demonstrations be given in towns and townships, that pamphlets in plain language be prepared by the health officer for general distribution, and especially that rules and suggestions, with reasons therefor, be placed in the homes of dairymen and dairy attendants.

DISEASES WHICH MAY RENDER MILK DANGEROUS.

TUBERCULOSIS.

This is probably the most important disease of cows from the standpoint of public health, and it is also the most prevalent. When Koch first discovered the cause of tuberculosis and combined the announcement of his discovery with the statement that he considered the affection identical in both man and cattle, this view was accepted by scientists as well as by the general public. His subsequent announcement in 1901, to the effect that this disease was different in man and in cattle, and that there was no practical need for preventing the use of the products of tuberculous animals for human food, was the cause of much rejoicing among those who were only too glad to grasp at any idea which would tend to separate the disease in man and in cattle, forgetting that bovine tuberculosis is also a dangerous disease to other cattle in the herd and should be stamped out for this reason aside from any danger to man.

As a result of this radical statement of Koch's, which was based upon incomplete and unsatisfactory evidence, several government commissions were appointed in different countries, and many private and public scientists immediately took it upon themselves to solve the question raised by that investigator. The results of these experiments were so strikingly similar that it is now the generally accepted opinion among scientists that people, especially children, may become infected with tuberculosis from cattle. It is not known to what extent such infection occurs, nor is it possible to obtain any definite percentage by the method formerly adopted of looking for the primary lesions in the intestinal canal, although much statistical evidence is recorded, showing that even by these figures primary intestinal tuberculosis of children has been observed in as high as 15.5 per cent of the tuberculous cases examined (Heller). Evidence which must be considered conclusive has been obtained by the Bureau of Animal Industry, as well as by Ravenel and a number of French investigators, showing that tuberculous infection may take place through the intestinal tract without leaving any lesion in the abdominal cavity, the first alteration being found in the lungs or the thoracic glands. Therefore the presence of pulmonary tuberculosis in infants without intestinal lesions is no indication that the disease was not transmitted by the food, and the statistics above referred to are thus shown to be below the true percentage of cases of tuberculosis of intestinal origin.

EVIDENCE OF TRANSMISSION FROM CATTLE TO PEOPLE.

These figures, however, do not give any satisfactory idea as to whether the bacilli entering the intestines originated from human or bovine sources. Owing to this fact it follows that the only way of determining the infection of people by bacilli of the bovine type is to study the lesions in the body of as many cases of human tuberculosis as possible. Already we have sufficient data to give us some idea of the extent of tuberculosis of the bovine type in children without considering the numerous cases of direct transmission recorded by many physicians, especially of instances of butchers and others receiving accidental infections of the skin with the bovine organism. Moreover, according to Von Behring, the question of infection in man usually goes back to childhood, as he believes that many of the cases of pulmonary tuberculosis in adults are of intestinal origin, infection having occurred primarily through the intestinal tract by drinking tuberculous milk during infancy and having remained latent until adult life. As vital statistics show that 14 out of every 100 people that die succumb to tuberculosis, while of the remaining 86 more than one-half show lesions of tuberculosis on post-mortem, although dying from some other cause, the foregoing statement of Von Behring is also practically pertinent in regard to the relation of human tuberculosis to the milk supply, especially in connection with the results of those investigators who have studied market milk and found from 2.7 to 55 per cent of the samples examined to contain tubercle bacilli.

Since direct experiments upon human beings are out of the question, the finding of the bovine type of tubercle bacillus in human lesions is the most direct and positive proof that tuberculosis of cattle is responsible for a certain amount of tuberculosis in the human family. Numerous experiments with this object in view have already proven this fact. Thus the German Commission on Tuberculosis examined 56 different cultures of tubercle bacilli of human origin and found 6 which were more virulent than is usual for human tubercle bacilli, causing marked lesions of tuberculosis in the cattle inoculated with them, and making over 10 per cent of the cases tested that were affected with a form of tuberculosis which, by Koch's own method, must be classified as of bovine origin. The bacilli, with the exception of a single group, were all derived from the bodies of children under 7 years of age, being taken from tubercular ulcers in the intestines, the mesenteric glands, or from the lungs.

In a similar series of tests conducted by the British Royal Commission on Tuberculosis, 60 cases of the disease in the human were tested, with the result that 14 cases were claimed by this commission to have been infected from bovine sources. Ravenel reports that of 5 cases of tuberculosis in children 2 received their infection from cattle. Theobald Smith has estimated that from 25 to 50 per cent of the cases of human tuberculosis starting in the cervical and mesenteric lymph glands are bovine in origin, while Park has recently found 4 cases of bovine infection out of 11 cases of generalized tuberculosis of infants, and 3 cases due to the bovine type of bacillus out of 16 cases of tubercular adenitis. Of 4 cases of generalized tuberculosis in children examined in the Biochemic Division of the Bureau of Animal Industry 2 were found to be affected with very virulent organisms, which warranted the conclusion that such children had been infected from a bovine source. The Pathological Division of the same Bureau has likewise, out of the 9 cases of infantile tuberculosis examined, obtained two cultures of tubercle bacilli that could not be differentiated from bovine cultures. In Europe so many similar instances of bovine tubercle bacilli having been recovered from human tissues are on record that it appears entirely proven that man is susceptible to tuberculosis caused by animal infections, and while the proportion of such cases can not be decided with even approximate accuracy, it is nevertheless incumbent upon us to recommend such measures as will guard against these sources of danger.[a]

MILK AS A CARRIER OF TUBERCULAR INFECTION.

The two principal sources of infection from cattle, and the only ones necessary to be considered, are the meat and milk of tuberculous animals. The fact that most of the cases of bovine tuberculosis above enumerated which occurred in the human occurred in infants points with grave suspicion to the milk rather than the meat supply. This naturally leads to the question of how and under what condition does the milk become dangerous, since Bang, Rabinowitsch and Kempner, Ernst, Ravenel, Smith, MacWeeney, Moussu, Gehrmann and Evans, Mohler, and many others have definitely determined the infectiveness of milk from tuberculous cows.

That milk coming from a tuberculous udder is capable of transmitting the infectious principle is conceded by all who have given the subject any consideration. It has been equally established that in advanced generalized tuberculosis the udder may secrete tubercle bacilli without showing any indication of being affected. Careful experiments performed by trained and eminently responsible investigators have also demonstrated beyond reasonable doubt that tubercle bacilli at certain times may be present in the milk of cows affected with tuberculosis to such a degree that the disease can be detected only by the tuberculin test, so that in a herd of cows in the various stages of tuberculosis it is to be expected that some of them will secrete tuberculous milk, which, when mixed with other cows' milk, makes the entire product dangerous.

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[a] Ravenel has collected the number of cases of human tuberculosis which have been studied with special reference to the type of bacillus causing them, whether human or bovine, and states that of the 306 cases reported, 63, or approximately 20 per cent, were due to the bovine tubercle bacillus.

In this connection it may be stated that the market milk of the District of Columbia has recently been examined by the writer for the presence of tubercle bacilli by the intra-abdominal inoculation of guinea pigs, and in 2 samples, or 2.7 per cent of the 73 specimens tested, virulent tubercle bacilli were recovered. The ease with which tubercle bacilli may be eliminated by the udder was strikingly illustrated by an experiment conducted by the Royal British Commission, in which a cow injected with human tubercle bacilli under the skin of the shoulder began excreting tubercle bacilli from the mammary gland seven days later, and continued to do so until its death from generalized tuberculosis thirty days after inoculation. Furthermore, Titze, of the Kaiserliche Gesundheitsamte, proved that human tubercle bacilli when injected into the jugular vein of milch cows may be excreted with the milk. In the first experiment the excretion of the bacilli began in the third week and continued until the 144th day. In a subsequent test tubercle bacilli began to be excreted after twenty-four hours, but no bacilli could be found after ninety-nine days. In both these cows only the milk from the left hind quarter proved to be infectious.

It has been shown by Gaffky and Eber in Germany and Schroeder in this country that, even when the tubercle bacilli are not being excreted by the udder, the dust and manure of the stable where the diseased animals are kept are in many cases contaminated with tubercle bacilli. This contaminated material may readily infect the milk during the process of milking, even though the milk comes from a healthy cow. The importance of this method of infecting milk can not be too greatly emphasized when it is known that cattle in prime condition, without any udder lesions and with but slight alterations in the lungs, frequently raise tuberculous mucus into the pharynx while coughing, then swallow this material and thus contaminate the feces. In a recent examination at the Bureau of Animal Industry Experiment Station of the manure passed by 12 cows just purchased from dairy farms supplying milk to the city of Washington and affected with tuberculosis to an extent demonstrable only by the tuberculin test, tubercle bacilli were found in over 41 per cent of the cases, both by microscopic examination and animal inoculations. The danger from this method of infecting milk is impressed upon us as consumers when we consider the prevalence of tuberculosis in dairy herds as disclosed by numerous tests.

PREVALENCE OF TUBERCULOSIS AMONG COWS SUPPLYING MILK TO THE DISTRICT OF COLUMBIA.

Judging from the results of recent tuberculin tests, it is believed that on an average between 15 and 25 per cent of all the cows which supply milk to the District of Columbia are tuberculous. During 1907 and the first half of 1908 the Bureau of Animal Industry supervised the testing of 2,468 cattle in 128 herds supplying milk to the District, with the result that 387, or 15.68 per cent, were found tuberculous. Many other tests have been made by local veterinarians of which the Bureau has no records. The percentage given is scarcely a fair estimate of the extent of tuberculosis in the dairy herds of this vicinity, since our tests include many herds which have either been cleaned previously by private tests or which have such a healthy appearance as to remove all suspicions of tuberculosis on a physical examination. Thus far these tests have all been voluntary on the part of the dairymen, and it is pleasing to note the large number who have had their herds cleaned of tuberculosis and the premises disinfected.

DANGER FROM TOXIN IN MILK OF TUBERCULOUS COWS.

Aside from the danger of tubercle bacilli in milk, some investigators (Le Blanc, Ripper, Jemma, and De Michele) consider the milk of tuberculous cows dangerous even when bacilli are not present, on account of the toxin it contains. Michellazzi has injected such milk into tuberculous animals and obtained a reaction.

ELIMINATE TUBERCULOUS CATTLE OR PASTEURIZE MILK.

To eliminate all tuberculous cattle from the herd or to pasteurize all milk coming from untested cattle should therefore be the object of all producers of milk, and sanitarians will be remiss in their whole duty should they neglect to guard against the products of tuberculous animals in their attempts to eradicate tuberculosis from man. This view was crystallized in a resolution adopted by the International Congress of Tuberculosis recently held in Washington, D. C., as follows:

> *Resolved*, That preventive measures be continued against bovine tuberculosis and that the possibility of the propagation of this infection to man be recognized.

TUBERCLE BACILLI IN OTHER DAIRY PRODUCTS.

Since milk is so often infected with tubercle bacilli, it is very evident that food products made from milk without submitting it to lethal temperatures during the process of their manufacture must frequently harbor virulent tubercle bacilli in undesirable numbers.

The investigations of Rabinowitsch, Klein, Laser, Bang, Petri, Dawson, Markl, Möller, and many others have conclusively shown that tubercle bacilli may be present in butter, buttermilk, margarin, and cheese when these products are offered for sale. Butter made in the customary manner and stored under the ordinary market conditions until time of sale, if dangerous through the presence of tubercle bacilli at the time of its manufacture, may retain its virulence through several months. This statement has been adequately proved by two series of experiments recently performed by the Bureau of Animal Industry.

In one series by Mohler, Washburn, and Rogers three samples of butter were tested. The first was made from milk to which bovine tubercle bacilli had been added just before churning. They were obtained from a luxuriantly growing culture upon glycerin bouillon. Ten centigrams were removed from the surface growth of the flask, carefully mixed in a sterilized solution, and added to 10 gallons of milk. The second sample was made from milk obtained from a cow affected with tuberculosis of the udder. In this milk tubercle bacilli of extreme virulence were present in great numbers. Both the first and second samples of butter were salted in the usual proportions of 1 ounce of salt to a pound of butter. The third sample was similar in every respect to the second, except that it was left unsalted. These samples of butter were tested upon guinea pigs, not only when first made, but also after storing for ten days in the ice chest, after holding in cold storage for sixty days, and again after retention in cold storage for a period of five months (one hundred and fifty-three days). The results showed that each of these samples harbored virulent tubercle bacilli throughout the entire storage period, and that at any time they were capable of infecting guinea pigs with tuberculosis if injected into the peritoneal cavity, and if the tuberculous butter was fed to the animals generalized cases of tuberculosis were still capable of being developed. In these experiments 10 guinea pigs were fed upon each butter sample for three consecutive days and 6 were inoculated with the same kind of material. Six weeks later they were chloroformed and the visceral organs of each were carefully scrutinized that every trace of tuberculosis might be detected. None of the lots of guinea pigs remained entirely free of tuberculosis, although those animals which were fed upon the contaminated butter failed to contract the disease as frequently as those which were injected. This experiment is to be extended further in order to determine the maximum time in which infected butter, both salted and unsalted, will remain virulent when kept in cold storage under normal trade conditions. As the temperature in the cold-storage room is very low, the evidence shows that the tubercle bacilli are held unchanged in the frozen butter for a long period, but that they slowly lose their vitality.

In another series of experiments by Schroeder and Cotton, of the Bureau Experiment Station, butter was made from the milk of a cow affected with udder tuberculosis. After salting at the rate of 1 ounce of salt to a pound of butter, the butter was kept without ice in a cellar in which the temperature remained fairly constant at 60° F., and from time to time, up to one hundred and sixty days from the making of the butter, guinea pigs were inoculated with portions of the butter. More than 60 guinea pigs were thus inoculated and, with the exception of 5 that died prematurely and 1 that was killed, all died of generalized tuberculosis, and the one that was killed was also found affected.

In cheese also tubercle bacilli may become mixed up with the curd during the process of manufacture, and they have been shown to remain virulent for over three months. As a result of Galtier's experiments conducted with cheese, both salted and not salted, which was found to contain tubercle bacilli when two months and ten days old he concluded that coagulated milk, fresh cheese, and salted cheese made from the milk of tuberculous cows may infect man, and that the by-products fed to swine and chickens may infect these animals. In experiments made in Switzerland to determine the fate of tubercle bacilli in cheese it was demonstrated that they died between the thirty-third and fortieth day in cheese made after the Emmental method, but considerably later in cheese made approximately after the Cheddar method. An emulsion of tubercle bacilli was added to milk at the same time as the rennet, and cheese was made from the milk in the manner required to obtain Cheddar cheese. From the time of manufacture average samples of the cheese were taken weekly, macerated in sterile water, and filtered. Guinea pigs were inoculated with portions of the filtrate, and it was found that the germinating power of the tubercle bacilli lasted one hundred and four days, but after one hundred and eleven days they were incapable of conveying the disease to guinea pigs by inoculation. Harrison concluded that these experiments justify the statement that Emmental cheese may be eaten with safety, as the period of ripening is much longer than the period during which the bacilli become inocuous. Cheddar cheese, he states, is seldom eaten under four months from time of manufacture, and during this period the tubercle bacilli lose their vitality. Notwithstanding this, however, the writer recommended the pasurization of the milk in order to make the cheese absolutely safe. a recent investigation conducted by the writer in cooperation with oane, tubercle bacilli have been demonstrated by guinea-pig inocutions in cheese one hundred and twenty-two days old, made after e Cheddar method.

In manufacturing margarin the method commonly employed is to bject the finely comminuted fat to a temperature not to exceed 50 . for one and one-half hours. Sour milk is then added and the hole mass is thoroughly mixed; dairy butter is next added, and a rtain proportion of oils (cotton, palm, cocoanut, etc.). Enough of e or more of these oils is added to lower the melting point to that f dairy butter. Hence it will be seen that artificial butter thus ade may be infected in three ways: First, from the fat secured rom the original cattle, as tubercle bacilli will withstand a temperaure of 50° C. for some hours; second, from the butter or soured ilk that has been added; and, third, from contamination during he course of its manufacture. Morgenroth made examinations of 20 amples of oleomargarin, purchased in the open market, and proved he presence of virulent tubercle bacilli in 9 of the specimens.

Other products which occasionally are consumed by people, but re used more extensively as food for live stock, will also serve to onvey tubercle bacilli from infected milk to those that are allowed o consume them. Thus whey from cheese factories and buttermilk nd separated milk from public creameries are all offenders in this respect and have been incriminated, especially in the feeding of hogs and calves.

VALUE OF THE TUBERCULIN TEST.

The symptoms of tuberculosis in cattle are not sufficiently prominent except in advanced stages or when superficially located to enable one to diagnose this disease by the ordinary methods of physical examination. And the cattle may, without showing any clinical symptoms, be in such a stage of tuberculosis as to render them capable of spreading disease. Indeed, an animal may be fat and sleek, eat and milk well, have a bright, glossy coat, and be apparently in the pink of condition, and still be passing tubercle bacilli through the feces or by an occasional cough, and thus endanger all the healthy cattle in the herd. Consequently, such adventitious aids to diagnosis as animal inoculation, biological test, serum agglutination reaction, and the tuberculin test are made use of in arriving at a definite opinion relative to the presence or absence of this disease. The value of all but the last of these is discounted by the technique required and their impracticability, while the tuberculin test is most satisfactory and is the best diagnostic agent known for the disease.

THE ORIGIN OF THE TUBERCULIN TEST.

Tuberculin was invented by Koch in 1890, and was first used experimentally in treating tuberculosis in man. In these cases it wa observed that its injection was followed by a rise of temperature which led veterinarians to apply tuberculin to suspected animals t see if a similar reaction resulted. Numerous experiments showed thi to be the case, and since 1891 the use of tuberculin as a diagnosti agent for tuberculosis of cattle has been almost universally adopte in all parts of the civilized world. No one thinks of accepting tuberculin as an absolutely infallible agent, but it is immeasurably mor dependable than any other method that has ever been used.

THE NATURE AND APPLICATION OF TUBERCULIN.

Tuberculin is the sterilized and filtered glycerin extract of cultures of tubercle bacilli. It contains the cooked products of the growth of these bacilli, but not the bacilli themselves. Consequently, when this substance is injected under the skin of an animal it is absolutely unable to produce the disease, cause abortion, or otherwise injure the animal. In case the injected animal is normal there is no more effect upon the system than would be expected from the injection of sterile water. However, if the animal is tuberculous, a decided rise of temperature will follow the use of tuberculin.

In practice the tuberculin test is applied by first taking a sufficient number of temperatures, usually three, at intervals of two hours to ascertain the normal variation of temperature of the animal to be tested. The dose of tuberculin (which should always be specified on the label) is then injected hypodermically between 8 and 10 p. m. on the day of taking the preliminary temperatures.[a] On the following day the "after" temperatures are recorded every two hours, beginning at 6 a. m. and continuing until twenty hours following the injection.

THE RELIABILITY OF THE TUBERCULIN TEST.

As a result of this method an accurate diagnosis may be established in over 97 per cent of the cases tested. The relatively few failures in diagnosis are included among two classes of cattle. The first class contains those that are tuberculous but which do not react either because of the slight effect of an ordinary-sized dose of tuberculin on an advanced case of the disease with so much natural tuberculin already in the system, or on account of a previous test with tuberculin which produces a tolerance to this material lasting for about six weeks. The second class includes those that are not tuberculous but which show an elevation of temperature as a result of (*a*) advanced pregnancy; (*b*) the excitement of œstrum; (*c*) concurrent diseases, as inflammation of the lungs, intestines, uterus, udder, or other parts, abortion, retention of afterbirth, indigestion, etc.; (*d*) inclosure in a hot, stuffy stable, especially in summer, or exposure to cold drafts or rains, or (*e*) any change in the method of feeding, watering, or stabling of the animal during the test.

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[a] The ophthalmo-tuberculin test and the cuti-tuberculin test, as their names imply, consist in the application of the tuberculin to the eye and to the scarified skin of the animal to be tested. These methods will not be discussed at present, as they are still in the experimental stage.

Notwithstanding all these possibilities of error, the results of thousands of tests show that in less than 3 per cent of the cases tested do these failures actually occur. In the first class the chances of error are decidedly reduced by the skilled veterinarian by making careful physical examination and diagnosing these advanced cases, and by the injection of double or triple doses into all recently tested cattle, with the taking of the after temperatures beginning two hours following the injection and continuing hourly for twenty hours. In the second class errors are avoided by eliminating those cases from the test that are nearing parturition or are in heat, or show evidence of the previously mentioned diseases, or exhibit temperatures sufficiently high to make them unreliable for use as normal. Then, in reading after temperatures it is advisable not to recognize as a reaction an elevation of temperature less than 2° F., or one which at the same time does not go above 103.8° F., and the temperature reaction must likewise have the characteristic rainbow curve. (Those cases which approximate but do not reach this standard should be considered as suspicious and held for a retest six weeks later.) In addition, a satisfactory tuberculin must be used; also an accurate thermometer and a reliable syringe in order that a sufficient dose of tuberculin may be given. Finally, the number of apparent errors of the tuberculin test will be greatly diminished if a careful post-mortem examination is made, giving especial attention to the lymph glands.

This low percentage of failures being the case, cattle owners should welcome the tuberculin test not only for their own interest but for the welfare of the public as well. Where this method of diagnosing the disease has been adopted tuberculosis is gradually being eradicated, while it is spreading rapidly and becoming widely disseminated in those districts where the tuberculin test has not been employed. Without its use the disease can not be controlled and the cattle owner is confronted with serious and continuous losses; with its use the disease can be eradicated from the herd, a clean herd established, and the danger of its spread to man removed. Tuberculin may, therefore, be considered a most beneficial discovery for the stock raiser. Strange to say, many of these men have been incredulous, antagonistic, or prejudiced against the tuberculin test by misinterpreting published statements, by incorrect, unsubstantiated, or exaggerated reports, and by alleged injurious effects to healthy cattle.

Law [a] has clearly stated the question when he says:

> Many stock owners still entertain an ignorant and unwarranted dread of the tuberculin test. It is true that when recklessly used by ignorant and careless people it may be made a root of evil, yet as employed by the intelligent and careful expert it is not only perfectly safe, but it is the only known means of ascertaining approximately the actual number affected in a given herd. In most infected herds, living under what are in other respects good hygienic conditions, two-thirds or three-fourths are not to be detected without its aid, so that in clearing a herd from tuberculosis, and placing both herd and products above suspicion, the test becomes essential.
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> In skilled hands the tuberculin test will show at least nine-tenths of all cases of tuberculosis when other methods of diagnosis will not detect one-tenth.

It is perfectly natural that there should be objection to its use among those who are not acquainted with its method of preparation or its properties; but it is difficult to explain the antagonism of farmers who are familiar with the facts connected with the manufacture and use of tuberculin. Probably the most popular objection to tuberculin is that it is too searching, since it discovers cases in which the lesions are small and obscure. While this fact is admitted, it should also be borne in mind that such a small lesion to-day may break down and become widely disseminated in a relatively short period. Therefore any cow affected with tuberculosis, even to a slight degree, must be considered as dangerous not only to the other animals in the herd but also to the consumer of her products.

THE HARMLESSNESS OF TUBERCULIN.

Furthermore, tuberculin must be considered as harmless for healthy animals in view of the results revealed by numerous tests covering vast number of animals. And it has also been clearly demonstrated that tuberculin interferes in no way with the milking function in healthy cattle; neither in the quantity of milk nor in butter-fat value has any variation been detected.

Nocard and Leclainche [b] state:

> Direct experiments and observations collected by thousands show that the tuberculin injections have no unfavorable effect. With healthy animals the system is indifferent to the inoculation; with tuberculosis animals it causes only slight changes, which are not at all serious.

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[a] Text Book of Veterinary Medicine, vol. 1, pp. 458, 465. Ithaca, N. Y., 1902.

[b] Les Maladies Microbiennes des Animaux, vol. 2, p. 85. Paris, 1903.

DISPOSAL OF AND INDEMNITY FOR REACTING ANIMALS.

Most of the objections to tuberculin would probably be removed if some method of compensation for the reacting animals could be devised. Thus, in Pennsylvania, where tuberculosis is being eradicated with more success than in any other State, and where there are usually three times as many voluntary requests on file for the application of the test as can be made, all reacting animals are paid for by the State. As the suppression of tuberculosis is a public health measure, it would appear perfectly logical for the State governments to reimburse cattle owners appropriately for the animals condemned and slaughtered. Provision could be made to pay 70 per cent of the appraised value of the condemned animals, not to exceed $30 per head for common stock or $60 for registered stock. Such legislation should also include a requirement for the testing of all cattle coming into the State for dairy or breeding purposes.

All tuberculosis animals should be slaughtered in abattoirs having Federal inspection, and the money obtained from carcasses which are inspected and passed for food and from the hide and offal of those carcasses condemned as unfit for food should be applied as part payment on the indemnity for their respective owners. The payment of indemnity for tuberculosis animals is a good business policy and would do more toward making the tuberculin test popular with cattle owners than any other possible action. And as a corollary of the latter more testing would be performed and more tuberculous cattle would be discovered at the start, but the gradual suppression of the disease would soon be manifest, as has been noted in Pennsylvania and Denmark. Furthermore, as Stiles has mentioned, if tuberculosis can be eradicated from dairy herds with but slight loss to the owner, the increase in the price of milk would naturally be inhibited, and the children of poor families would consequently be in less danger of having this very important article of their diet decreased.

CONCLUSIONS REGARDING THE TUBERCULIN TEST.

As a result of the careful study of the tuberculin test Salmon[a] draws the following conclusions:

1. That the tuberculin test is a wonderfully accurate method of determining whether an animal is affected with tuberculosis.
2. That by the use of tuberculin the animals diseased with tuberculosis may be detected and removed from the herd, thereby eradicating the disease.
3. That tuberculin has no injurious effect upon healthy cattle.
4. That the comparatively small number of cattle which have aborted, suffered in health, or fallen off in condition after the tuberculin test were either

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[a] Yearbook of the United States Department of Agriculture, 1901, p. 592.

diseased before the test was made or were affected by some cause other than the tuberculin.

SUMMARY OF DIRECTIONS FOR MAKING THE TUBERCULIN TEST.

1. Stable cattle under usual conditions and among usual surroundings, feeding and watering in the customary manner.

2. Make a physical examination of each animal, and give to each one some designation by which the animal will be known throughout the test.

3. Take each animal's temperature at least three times at two or three hour intervals on the day of injection; for instance, at 2, 5, and 8 p. m.

4. At 8 or 10 p. m. inject a dose of tuberculin under the skin in the region of the shoulder, using a sterile hypodermic syringe after disinfecting the skin at the seat of injection with a 5 per cent solution of carbolic acid or a similar antiseptic solution.

5. Tuberculin is not always concentrated to the same degree, and therefore the dose, which should always appear on the label, varies considerably. The dose of imported tuberculin is 0.25 c. c. for an adult cow, and before injection is diluted with sterile water to 2 c. c. The tuberculin made by the Bureau of Animal Industry is prepared so that it will not be necessary to dilute it, and the dose is 2 c. c. for an adult animal. Yearlings and 2-year-olds, according to size, should receive from 1 to 1½ c. c., while bulls and very large animals may receive 3 c. c.

6. At 6 a. m. on the day following the injection of tuberculin commence taking temperatures, and continue every two or three hours until the twentieth hour after injection, at which time if there is no tendency for the temperature to rise the test may cease.

7. A rise of 2° F. or more above the maximum temperature observed on the previous day, providing the temperature after injection exceeds 103.8° F., should be regarded as an indication of tuberculosis. Those cases which approximate but do not reach this standard should be considered as suspicious, and held for a retest six weeks later, giving double the original dose.

FOOT-AND-MOUTH DISEASE.

The recent outbreak of foot-and-mouth disease in Michigan, New York, Pennsylvania, and Maryland, which started October 16, 1908, has been the occasion of many inquiries regarding the effect of this disease upon the milk supply and the danger of its transmission to man. A brief discussion of these subjects at this time would therefore seem appropriate.

Foot-and-mouth disease, also known as "aphthous fever," "epizootic aphtha," and "eczema contagiosa," is an acute, highly infectious disease of cattle and other domestic animals the characteristic feature of which is the eruption of vesicles on the mucous membrane of the mouth and on the skin between the toes and above the hoofs. The vesicles rupture, forming erosions and ulcerations accompanied by salivation, great tenderness of the affected parts, loss of appetite, lameness, emaciation, and diminution in the quantity of milk secreted.

In the early stages or in a mild attack of the disease the milk may present only a few abnormal characteristics, such as a fall in its specific gravity to 1023–1025, and the reduction in the quantity of sugar and casein. When the disease is fully developed, or about the third day, the milk invariably contains inflammatory products of a very pronounced character, and the quantity of milk secreted is greatly reduced. Cows affected with the malignant form of the disease lose practically all of their milk, but if the disease is mild in character the decrease will be from one-third to one-half of the usual yield.

The milk becomes thinner, bluish, and poor in fat. Only in rare cases does the fat content increase with the diminution of the milk secretion. The casein and sugar content is reduced, but the salt content is increased and the acidity diminished. If the udder becomes involved the milk has a slimy consistence and is yellowish and viscous like colostrum. It frequently contains coagulated fibrin and blood, so that a considerable sediment forms after standing, while the layer of cream which rises is thin and of a dirty color. Occasionally no layer of cream is formed, but the milk appears as a uniform, slimy mass of a bad odor, and a repulsive, rancid taste. This slimy consistence is due to the large quantity of albumen and globulin contained. The sediment contains leucocytes, desquamated epithelial, and broken-down tissue cells in large quantities, besides fibrin and red blood cells.

This affection is transmissible to man through the ingestion of raw milk, buttermilk, butter, cheese, and whey from diseased animals, and also directly, though more rarely, from the saliva, secretions, or other infected material which may gain entrance through the abrasions of the skin. Children are not infrequently infected by drinking unboiled milk during the periods in which the disease is prevalent in the neighborhood, while those persons in charge of diseased animals become infected through contact with the diseased parts as by milking, slaughtering, or caring for them. In such cases the symptoms resemble those observed in animals. There is fever sometimes with vomiting, painful swallowing, heat and dryness of the mouth, followed by an eruption of vesicles on the buccal mucous membrane and very rarely by similar ones on the fingers. These vesicles appear on the lips, gums, cheek, and edge of the tongue, and are about the size of a pea. The vesicles soon rupture, leaving a small erosion which is soon covered by a thin crust under which the new formation of epithelium proceeds rapidly. The skin eruption mostly appears on the hands, tips of the fingers, base of the nails, volar surfaces of the finger tips, and more seldom on the toes and other parts of the body. Besides these local changes, during the course of the disease there are occasionally observed headache, pain in the limbs, vertigo, abdominal cramps, vomiting, diarrhea, and weakness. The disease is very seldom fatal, usually appearing in a very mild form except in weakened children in whom an accompanying intestinal catarrh may lead to a fatal termination. Those veterinarians who have had considerable experience with this disease among animals regard the human affection as by no means uncommon in countries where foot-and-mouth disease prevails, but the disturbance of health is usually too slight to come to the notice of the family physician. The frequency of such infection is established by numerous observations which have been recorded in the literature of foot-and-mouth disease, showing that human infection has constantly accompanied the outbreaks among cattle. Valentin was probably the first to suggest the infectiousness of the milk and the transmissibility of the virus to man through the milk of diseased animals. He reported several cases where people became infected during the outbreak in Hesse in 1695. Sagar, in 1765, reported a similar infection of men in Moravia from drinking infected milk. In 1778 all the residents of an Austrian monastery developed a vesicular eruption in the mouth after drinking virulent milk. Hertwig, Mann, and Villain conducted an experiment on themselves in 1834 by drinking milk warm from a cow which was suffering from an attack of foot-and-mouth disease. Five days later vesicles appeared on the hands, fingers, tongue, cheeks, and lips of Hertwig, while the eruption in the other two was confined to the buccal mucous membrane.

Allbutt observed the vesicular eruption in the mouths of three children in Yorkshire during the English outbreak in 1883, and obtained information of a number of similar cases in the community. During the 1893 outbreak in Germany a shepherd infected himself by holding in his mouth a knife which had been used in paring the diseased feet of his sheep. A number of milkmaids were infected through milking, the vesicles appearing principally on their hands. A child fed on unboiled milk of affected cows developed an eruption of blisters on the tongue, lips, and soft skin between the fingers and toes. Furthermore, in the Berlin outbreak of 1895 a number of those who drank infected milk developed fever, followed by the formation of vesicles on the tongue and lips. The acute disease lasted about five days, leaving a feeling of great weakness for some time. Virchow made an investigation of these cases and unhesitatingly pronounced the affection to be foot-and-mouth disease. A collection of the cases of transmission of this disease to man through the consumption of milk has been prepared by Würzburg, while the work of Brussenius & Siegel contains a full bibliography of the literature of such transmission up to 1896. Similar cases of infection resulting from the use of butter made from infected milk are on record, while Schneider mentions instances where human infection followed the consumption of infected cheese. Similarly, Freidberger & Fröhner record cases which were caused by infected buttermilk.

There have been but few outbreaks of the disease in the United States, and therefore recorded cases of its transmission to man in this country are quite rare. Law reports having observed the disease in man from drinking infected milk during the epizootic of 1870 in the Eastern States, but the outbreaks of 1880 and 1884 affected such a small number of animals and was so quickly suppressed that no instance of its transmission to man was recorded. A few cases have been reported by Brush[a] accompanying the New England outbreak of 1902. Similar reports have likewise been received regarding the appearance of vesicular eruptions in the mouths of children during the 1908 outbreak, and the history of these cases incriminates the milk consumed. In both of these last outbreaks the sale of milk was stopped as soon as the disease was found among cattle, and therefore the opportunity for the infection of man was not so good as when the disease is more widespread, affecting practically all the cattle of the country. In some instances, however, the people used the milk after pasteurization, thus avoiding the possibility of infection, or the milk was discarded after having been rendered harmless by the addition of formalin.

Foot-and-mouth disease has only made its appearance in the United States on the above-mentioned occasions, having always been introduced from some foreign country. Its spread among cattle is very rapid, owing to the highly infectious character of the virus, but fortunately every outbreak upon American soil has thus far been quickly followed by its total suppression.

The method of eradicating the outbreaks in 1902 and 1908 consisted in the rigid quarantine of all infected premises and the animals upon them, in slaughtering the diseased and exposed animals at the earliest practicable moment, and in thoroughly disinfecting the stables and the contents of the buildings in which they had been sheltered. In this manner the disease was confined in both outbreaks to but four States and was completely eradicated in a comparatively short time in each instance.

The causative agent of this disease has not been isolated, although numerous attempts have been made to cultivate and stain it. The experiments of Loeffler & Frosch in 1898 have shown that the virus will pass through the finest Chamberland filter, thus indicating its ultramicroscopic size and the reason it has not been detected by staining methods. They inoculated experiment animals with this filtrate free from all indications of bacterial growth and transmitted the disease through a series of animals. These results have since been confirmed by other investigators. Observations which have been made upon the viability of this filterable virus led to the conclusion that the contagion is quite readily destroyed, and milk which has been pasteurized for the elimination of tubercle and typhoid bacilli will not prove capable of transmitting the disease to persons or animals fed with it. Experiments which have been made in recent years proved that the contagion will lose its virulence after fifteen minutes exposure at 50° C., by being heated for ten minutes at 70° C., while exposure to 100° C. destroys it at once.

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[a] Journal of American Medical Association, vol. 40, p. 1700, June 20, 1903.

The following disinfectants will destroy the virus in one hour: Ordinary whitewash; 1 per cent solution of carbolic acid; 3 per cent solution of soda, and 1 per cent solution of muriatic acid.

On the other hand, the resistance of the virus to the influence of low temperatures is quite marked, and the infected lymph will retain its activity for at least a month when placed in a refrigerator, while exposure to a temperature of minus 48° C. for about three hours did not destroy its virulence. Infectious milk three or four days old, after having turned sour, or milk to which rennet has been added, is not capable of transmitting the contagion In fresh cattle or swine manure the infectious material is very soon destroyod at a depth of over 1 foot, owing to the heat developing therein in consequence of decomposition and reaching about 70° C.

The experiments which have been made in Denmark and Germany indicate that the form of pasteurization recommended in article 20 of this bulletin is undoubtedly sufficient to kill the infectious principle of foot-and-mouth disease.

ACTINOMYCOSIS.

This disease, while not at all infrequent in the maxillary regions of cattle, is quite rarely located in the udder. It is readily mistaken for tuberculosis, owing to the diffuse lesions and the character of the pus. While no known case of actinomycosis in man has been traced to the milk, it is nevertheless advisable to condemn the milk from an infected udder, especially since the virus of the disease in man, in most cases, has been found to enter the body through the alimentary canal. Furthermore, there is usually in actinomycosis a mixed infection with pus-producing cocci, which emphasizes the necessity for prohibiting the use of the milk from such udders.

BOTRYOMYCOSIS.

Botryomycosis of the udder is only occasionally met with, but when it is observed the utilization of the milk therefrom should not be permitted. The disease is chronic and is accompanied by new connective tissue formation and burrowing sinuses from which pus escapes. Mixed infection is liable to occur in this disease also, which adds to the danger of consuming the milk.

ANTHRAX.

In this disease the milk has an abnormal appearance and decomposes rapidly. The bacterium of anthrax has been recovered from milk fourteen days after it had been taken from an infected cow, which illustrates the importance of prohibiting the use of milk from such animals.

COWPOX.

This disease, which is probably becoming more common in this country, renders the milk unfit for food, and its distribution from cows so affected should not be permitted, inasmuch as the milk may become contaminated from the pustules and ulcers on the teats and in the sinuses of the udder, and produce infection by the alimentary canal of young children if it is consumed in a raw state.

The appearance of dark-brown crusts on the teats and udders of cattle is suggestive of several conditions and should be carefully examined, particularly since the isolation by Dean and Todd of an organism identical with the Klebs-Loeffler bacillus from such lesions as well as from the milk. Diphtheria is not a disease of cattle, but it is possible for an abrasion to become infected with this organism from a human origin and the local lesion to spread until it involves the milk sinuses. It was also suggested that this udder lesion might be due to an infected milker following the all too common habit of spitting on his hands before commencing to milk, and the bacillus passing up the milk duct might thereby infect the sinus.

RABIES.

The virus of rabies has in several instances been reported to have been passed to the offspring through the mother's milk. While it is not probable that cattle would be milked after the symptoms of rabies developed, it is nevertheless important to realize the danger of using such milk and the necessity for preventing calves from sucking such diseased cows.

MAMMITIS, MASTITIS, OR GARGET.

This disease or series of diseases of the udder is by far the most frequent alteration noted. Usually only one quarter is affected, although the whole udder may at times be involved. The affected parts are greatly swollen and more or less painful in the early stages. The milk, at first normal in appearance, soon changes its character, becoming watery, light brown in color, and in some cases contains flocculi and pus cells, and appears tenacious, slimy, or ropy. The cause of this condition is usually a streptococcus, although staphylococci are frequently incriminated in suppurative conditions of the udder, especially where abcess formation occurs. The milk from such an udder is objectionable from an esthetic standpoint and is also liable to give rise to gastro-intestinal disorders, especially in children. Such milk should be prohibited until the inflammatory condition entirely disappears.

LEUCOCYTES IN MILK.

The number of leucocytes in milk and their significance are receiving the serious attention of those bacteriologists who are striving for a purer milk supply. The question as to what number of leucocytes should be regarded as abnormal is still the subject of investigation, but the opinions of contemporaneous workers are becoming more uniform as the methods for the determination of these leucocytes are reaching greater perfection. Previously it was considered that but few leucocytes were contained in the milk of healthy cows, and when a certain increased number of leucocytes were observed in milk it was suggestive of inflammation of the udder, termed mammitis or garget. As the milk in this disease may contain pus without changing the appearance of the product, such milk may readily be accepted by the consumer as normal. It is therefore of importance to be able to designate the cows and the milk so affected, especially as the causative agents of this condition are micrococci, which likewise produce intestinal disorders when consumed by infants. Even when the udder inflammation is slight an increase in the number of leucocytes secreted in the milk may be observed, and it was the recognition of this fact which has caused the introduction of microscopic examination of the milk for the determination of the presence or absence of garget. However, with the technique at present employed in the numerical determination of leucocytes there is too narrow a margin between the leucocytes found in the milk of healthy and those in diseased cows to make this form of diagnosis satisfactory and practicable. This is particularly true of milk from healthy cows during the first week of lactation, although at this time the normal increase of leucocytes would be accompanied by colostrum corpuscles. While the several methods recommended by different investigators are not directly comparable, it is nevertheless evident that an entire lack of harmony exists at present among them which makes the reliability of one or more of the methods at least doubtful. The Doane-Buckley and Frommsdorff methods are probably the most preferable, but neither of these is perfect and should not be depended on per se for the determination of udder inflammations by the examination of market milk or even the mixed milk of a herd.

Recently Russell and Hoffmann[a] have presented an improvement in the technique for determination of the leucocyte content of milk. Their experiments show that many leucocytes are not recovered in the sediment after centrifugalization by the usual methods, and that it is necessary to heat the milk to a point where the creaming power is so greatly diminished as to alter the physical arrangement of the fat globules. In this manner practically all the leucocytes which were enmeshed are liberated and thus enabled to be thrown down and recovered in the sediment. These observers used the Doane-Buckley quantitative method with milk which had been heated momentarily at 70° C. and obtained a count which was in some cases as much as four times greater than that secured from the same lot of milk unheated. This increase is usually so marked that it is far beyond the limits of analytical error, and, furthermore, such error is reduced to a minimum by this method of determination. These experiments have opened up an interesting line of investigation, and the limits of lactic leucocytosis, which have been adopted by some States, based upon the old standards, must be modified accordingly. Since it has been found that the heating of milk is absolutely essential in obtaining a somewhat definite idea of the number of leucocytes in milk, and inasmuch as this improvement in the technique, which is as necessary as any other part of the process, results in a much higher count than has heretofore been observed in any of the previously described methods, it is evident that there must be a higher leucocyte standard applied for judging milk than has obtained in the past. Milk inspection may be greatly benefited by the establishment of some rational standard for the leucocyte content of milk, but more study must be given to this subject in order to obtain the desired knowledge.

GASTRO-ENTERITIS.

The milk of cows affected with gastro-enteritis is of an abnormal character, being watery, of bitter taste, and changes quickly to a "sweet curdle." This milk is liable to produce digestive disturbances in the consumer and should not be utilized.

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[a] American Journal of Public Hygiene, p. 285, August, 1908.

MILK SICKNESS.

A rather peculiar disease, called "milk sickness," is found in the central part of the United States, where it at times occurs as an epidemic among cattle and people. In cattle the first indication of disease is dullness, followed by violent trembling and great weakness, which increases during the succeeding day until the animal becomes paralyzed and dies. Through the ingestion of flesh, milk, or dairy products of an affected animal the disease is transmitted to man or to another animal, and attacks produced in this way most frequently prove fatal. In man the disease develops with marked weariness, vomiting, retching, and insatiable thirst. Respirations become labored, peristalsis ceases, the temperature is subnormal, and the patient becomes apathetic. Paralysis gradually follows and death takes place quietly without rigor mortis.

Many efforts have been made to elucidate the question regarding the nature and cause of this disease, but although many theories have been discussed none of them has so far been generally accepted. Some investigators hold that the disease is of micro-organismal origin, some that it is due to autointoxication, while others think it is caused by vegetable or mineral poisons. All seem to agree, however, that the disease is limited to low, swampy, uncultivated land, and that the area of the places where it occurs is often restricted to one or a few acres. Furthermore, when such land or pastures have been cultivated and drained the disease disappears completely.

The discovery of a new focus of this disease in the Pecos Valley of New Mexico in November, 1907, gave Jordan and Harris[a] the opportunity of studying this peculiar affection by modern bacteriological methods. As a result they have succeeded in isolating in pure cultures from the blood and organs of animals dead of this disease a spore-forming bacillus which they name "*Bacillus lactimorbi*." With this bacillus they have reproduced in experiment animals the symptoms and lesions peculiar to milk sickness or trembles, and from these animals the same organism has been recovered in purity. It therefore appears to have been demonstrated that the bacillus in question is the actual cause of the disease. As Jordan and Harris have already indicated, more comprehensive studies, based on a larger supply of material, are desirable in order that the many obscure and mystifying features connected with the etiology of this rapidly disappearing disease may be elucidated.

From the above facts it seems evident that milk sickness is an infectious disease communicable to man, and the cattle owners should therefore not be permitted to make use of the meat or milk of affected animals for human consumption.[a]

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[a] The Journal of the American Medical Association, Vol. L, No. 21, May 23, 1908.

SEPTIC OR FEBRILE CONDITIONS.

The presence in the dairy of cows affected with such septic conditions as puerperal sepsis, septic metritis, diffuse phlegmon, suppurative wounds, and extensive ulcerations constitutes a grave danger to the milk supply, inasmuch as the milk may become infected with the pus-producing organisms, among which the streptococci are capable of causing enteritis in man. The milk of cows suffering from febrile conditions, especially when associated with sepsis, should also be excluded. In the case of small single wounds which cease to suppurate, the milk may be used without danger, providing the teats and udder are well cleaned before each milking.

ABNORMAL APPEARANCE AND CONDITIONS OF MILK.

The udder acts as a natural emunctory, like the kidney, and in consequence of its natural selective powers certain active principles contained in various foods, drugs, and poisons are eliminated thereby.

SLIMY, STRINGY, OR ROPY MILK.

These conditions of the milk are not an uncommon occurrence and sometimes are produced by a diseased condition of the udder of the cow, although in the majority of cases these abnormal appearances of milk are caused by various kinds of bacteria infecting the milk after it has left the udder. While this altered milk may be perfectly wholesome, it is nevertheless unpalatable, and most consumers in this country would rather do without than drink such material.

BITTER MILK.

This condition in the milk is second in importance only to the slimy milk and causes much trouble to the dairyman. Bitter milk may originate from two different sources. The first source is dependent upon the cow, while the second is due to the growth of bacteria in the milk after it has been drawn from the animal. The difference between these two classes of bitter milk is that the first has a decidedly bitter taste when freshly drawn, while the second class is sweet when taken from the cow, but the bitterness occurs after standing for a short time and increases in intensity. Only the former will be dealt with here. Bitter milk, when produced in the cow, may result from improper feeding with such herbs as lupines, wormwood, etc., or with raw Swedish turnips, cabbages, etc. Bitter milk may also be observed during the late stage of lactation and has followed the infection of teat ducts with bacteria which act on the proteids as an enzyme, converting them into peptones and other products to which the bitter taste is probably due.

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[a] For further discussion of this subject see article No. 6, this bulletin, by Dr. G. W. McCoy.

COLORED MILK.

Red milk may be produced by the effects of bacteria, but is usually the result of mixture of blood with the milk, due to an abrasion of the udder or teats or to some other traumatism of the udder. It may also be due to the cow eating material containing a large amount of silica, as sedges, rushes, etc., or to plants containing red pigment, as madder root. Other plants which are said to impart color to milk are alkanet, field horsetail, meadow saffron, and knot grass. *Bacillus cyanogenes*, the cause of blue milk, at times gets into the udder through the milk ducts and leads to a bluish discoloration of the secretion.

TASTE AND ODOR.

The flavor of milk is very readily affected by the character of the feed, as, for instance, by turnips, garlic, wild onions, moldy hay and grain, damaged ensilage, and distillery grain. The latter is said to cause hyperacidity of the urine and consequent eczema. With proper precautions, however, these substances can be fed to dairy cattle without producing ill effects in the milk. The deleterious substances excreted with the milk are usually volatile oils contained in the food. They are found in the milk as well as in the body, generally in the largest quantity during the digestion of the food containing them, being eliminated rapidly through the various excretory channels. Thus, if these substances are fed eight or ten hours before milking, or if cattle in the spring are removed from the pastures containing garlic this length of time before milking, there will be little or no danger of contaminating the milk. Overkept, fermented, and soured feeds tend to produce acidity and other changes in the milk. Swill, spoiled gluten meal, and ensilage put up too green are all more or less injurious to milk. Distillery swill, in addition to the bad flavor which it gives the milk, may cause the secretion of small quantities of alcohol in the fluid. That such alcoholic milk is deleterious to children as well as to the calves and lambs fed on it is a well-known and accepted fact.

Milk is also modified very sensibly by the use of certain medicines, and the list of drugs which are excreted in the milk and give it an abnormal odor or flavor or render it deleterious to the consumer is uite lengthy. Among the more important may be mentioned opium, ll volatile oils, purgative salts, rhubarb, arsenic, mercury, lead, zinc, ron, creolin, scammony, iodin, potassium iodid, antimony, bismuth, ammonia, and certain acids.

POISONOUS MILK.

Toxic properties may be manifested in the milk of cows that have eaten certain poisonous plants. Thus poison ivy (*Rhus toxicodendron*) produces a condition in cattle during which the milk is capable of producing in the consumer severe gastro-intestinal symptoms with weakness. Leaves of the common artichoke are also said to produce certain toxic properties in the milk which result in abdominal pains and diarrhea in the person consuming it.

COLOSTRUM.

Milk should not be used within fifteen days of parturition or during the first five days after parturition. All cows should be dried off at least fifteen days before calving, not only for the sake of the animal, but also on account of the poor quality of such milk at that time. This milk before and after parturition is called "colostrum," and is a yellow, viscid fluid of a strong odor, bitter taste, and acid reaction. The ingestion of such milk is liable to produce diarrhea, colic, and other digestive disturbances.

RECOMMENDATIONS.

In view of the facts above enumerated the following recommendations are made as a basis for laws and for regulations by public health officers:

1. That all cows on dairy farms producing milk for market purposes be tagged, tattooed, or otherwise marked for identification.
2. That all milk produced on such dairy farms shall either come from tuberculin tested cattle, which shall be retested at least once a year, or be subjected to pasteurization under the supervision of the health authorities in case the herd is not tuberculin tested.
3. That no additions to any herd, whether the herd has been tested or not, shall be made in the future without subjecting the additional cattle to the tuberculin test.
4. That no license for the sale of milk shall in future be granted except to applicants having herds free of tuberculosis.
5. That the milk of cattle showing any of the udder affections above mentioned, or anthrax, rabies, gastro enteritis, septic conditions, or clinical symptoms of tuberculosis, shall not be utilized as human food, even though the milk be pasteurized. Milk from cows

fifteen days before and five days after parturition and that from animals receiving any of the deleterious medicaments or foodstuffs previously mentioned shall likewise be excluded.

6. That veterinary inspectors of the health department make frequent visits to dairies having untested herds, in order that they may discover all advanced cases of tuberculosis, or udder tuberculosis, as early as possible.

7. That the various States pass laws granting an appropriate indemnity to all owners of tuberculous cattle which come under their respective jurisdiction, the said animals to be slaughtered in abattoirs having Federal or other efficient inspection.

15. THE RELATION OF THE TUBERCULOUS COW TO PUBLIC HEALTH.

THE RELATION OF THE TUBERCULOUS COW TO PUBLIC HEALTH.

By E. C. SCHROEDER.

Superintendent Experiment Station, Bureau of Animal Industry, Department of Agriculture.

Under the conditions of our present civilization the dairy cow fills a unique place: her living body is the source of the most important of all human foods; she has become an essential factor among our modern institutions; remove her and either a substitute must be found or many thousands of young children will die of starvation. The woman who can feed her infant at her own breast until it is old enough to thrive without milk is nearer the exception than the rule, so that either the cow or some other milk-producing animal must, as a sheer necessity, be available to serve the purposes of a human foster mother. After children have passed the period during which milk is a requisite article of food, most of them continue its use as a beverage and add butter to their diet as a second product from the cow. Later on cream and cheese are added, and the use of milk to some extent as a beverage, and of cream, butter, and cheese as regular, current articles of food, is continued to the end of life. Hence, even if we are not greatly influenced by the idea that it is disgusting and barbarous to eat substances that are obtained from the living bodies of diseased cows, we must feel that it is important to make a careful inquiry regarding the transmissibility to ourselves, through the use of dairy products, of the commonest disease with which dairy cows are affected. The need for this inquiry is emphasized by the knowledge that the commonest and most important disease of cows is also the commonest and most important disease of mankind, and by the fact that though the disease in question, tuberculosis, is one of the few infectious diseases to which widely different species of animals are susceptible, its commonest victims are persons and dairy cows.

The indispensable cause of tuberculosis is the multiplication of tubercle bacilli in the animal body. Bacilli do not grow and multiply in animal bodies until they have been introduced into them from without, and tubercle bacilli grow and multiply nowhere else in nature. The propagation of tuberculosis therefore depends upon the tubercle bacilli that emanate from the bodies of tuberculous individuals, human and animal, and the widespread and common occurrence of tuberculosis is due to the unguarded and dangerous expulsion and dissemination of tubercle bacilli by the victims of tuberculosis. This is the basis for the practically unanimous conclusion among those who are informed on the subject, that in our fight for the suppression and eventual eradication of tuberculosis we must strive to control and make harmless all the sources from which tubercle bacilli are scattered.

As persons and dairy cows are the commonest subjects of tuberculosis they are also the commonest sources from which tubercle bacilli emanate, and as the exposure of persons to persons through the ordinary routine of life, and the exposure of persons to dairy cows through the lifelong use of dairy products, are more direct and intimate than the exposure of persons to other possible sources of tuberculous infection, we may conclude that the two most important sources of tubercle bacilli against which public health must seek to defend itself are tuberculous persons and tuberculous dairy cows. Of these two sources the former is probably the more important, but only little can be said about it here, as the latter is the subject of this article, and the little that is permissible must be limited to the infection of dairy products when they are exposed to tuberculous or consumptive persons.

Persons affected with tuberculosis of the respiratory passages, the lung, throat, etc., expel tubercle bacilli with their sputum and with the particles of fluid sprayed from their mouths and noses during accelerated expiratory acts. Such persons are not necessarily dangerous to public health when they observe a number of simple precautions relative to the disposition of the infectious material they expel from their bodies, but they can not keep their environment sufficiently free from tubercle bacilli to make it a safe place for the exposure of food that is to be eaten by others. Dairy products are usually eaten in a raw state; that is, without previous exposure to a germicidal process like cooking, and hence it is especially desirable that they should not be handled by, and should not be exposed in the environment of, tuberculous persons.

The expulsion of tubercle bacilli by those who are affected with tuberculosis and the mode of its occurrence justify the enforcement of health regulations that will exclude all tuberculous persons from serving in occupations like food vendors, cooks, waiters, milkers, creamery employees, butter makers, etc.

A clear conception of the danger to which public health is exposed through the use of food products derived from tuberculous dairy herds requires that we should have some knowledge on the following, special subjects, which will be discussed in order: The character of tuberculosis as a disease of cattle; the manner in which tubercle bacilli are expelled by tuberculous cattle; the general appearance of tuberculous cattle that expel tubercle bacilli; how tubercle bacilli from cattle get into milk and other dairy products; the virulence and vitality of tubercle bacilli in dairy products; the proportion of tuberculous cows among those in use for dairy purposes; and, finally, the frequency with which dairy products have been proven under existing conditions to contain tubercle bacilli.

THE CHARACTER OF TUBERCULOSIS AS A DISEASE OF CATTLE.

Tuberculosis of cattle, as of persons, may be acute and rapidly progressive and run its course quickly from infection to death. This is rare. As a rule, it is insidious, chronic, and slowly progressive, and the bodies of its victims are able to adapt or adjust themselves to the gradually increasing, destructive changes it causes until quite extensive harm has been done or vitally important organs have become seriously involved. The result is that the disease may be present in the body a long time without external manifestations of its existence. It may attack any part of the body singly and remain confined to it or it may attack several or many parts simultaneously or successively. Its favorite location in the bodies of cattle, as in those of persons, is the lung.

The tuberculous lungs of cattle do not show the decided cavity formation seen in human tuberculous lungs, but cavities in direct communication with the exterior of the body through bronchial tubes, more or less completely surrounded by heavy, dense, connective-tissue walls, are common. The dissimilarity of the lesions in the lungs of persons and cattle are no doubt the result of normal, anatomical differences. The lungs of cattle, unlike those of persons, have very decidedly outlined lobules, which are separated from each other by webs of loose, elastic, connective tissue. This tissue is so abundant that it admits of a serous and cellular infiltration through which the lobules may be separated from each other as much as a sixteenth or even an eighth of an inch, and consequently not only pulmonary tuberculosis, but most lung diseases of cattle and other ruminants, have a special, distinct character.

Tuberculosis is more effectually concealed in the bodies of cattle than in those of persons, and we all know how long a diagnosis with persons may remain in doubt. Perception, or the faculty of receiving impressions, is keenest where the nervous system has the highest development. Persons, though their perception of pain is much keener than that of lower animals, complain little during the earlier stages of tuberculosis, because, we may assume, they experience little pain or distress. Cattle, with their lower perception and comparatively insignificant means to express suffering, do not complain at all because of the pain and distress tuberculosis causes them.

The frequency with which tuberculous subjects cough depends largely upon perception, or sensations in their lungs and throats that prompt them to cough. Cattle, shown on autopsy to have extensive, advanced, tuberculous lesions of the lung, though observed long periods of time before their death, were found to cough only a little more frequently than cattle shown on autopsy to have healthy lungs. When tuberculous cattle cough it is usually a single, accelerated expiratory effort, or at most two or three such efforts in succession, which is sufficient to raise the material that has accumulated in their larger air passages far enough into their mouths to be swallowed. Expectoration, which is common with persons, does not occur, and paroxysms of sustained coughing, also common with persons, are very rare and occur only during the last stages of pulmonary tuberculosis.

The bodies of all animals are stronger and more capable, as a whole as well as in their individual parts, than the ordinary vicissitudes of life require them to be. The difference between the actual strength and capability and that ordinarily required is known as the factor of safety. The rarely interrupted, placid routine of a cow's life enables her to derive full benefit from the factors of safety in her body when she becomes affected with a slow, chronic disease like tuberculosis, the lesions of which are circumscribed in the sense that they do not seriously affect parts of the body remote to those in which they are located; hence the factors of safety greatly help to prevent tuberculous disease in the bodies of cows from showing itself by external symptoms. An example of this is seen in illustration No. 15, which shows a cow affected with advanced tuberculosis.

The tuberculous disease of the cow represented in the picture is partly located in the glands of her throat; the enlarged glands press on and narrow the passage through which air reaches her lung to such an extent that she almost dies of suffocation when she is driven a short distance at a moderately fast walk. The general condition of the cow shows that the opening through which the air she breathes must pass is still large enough for ordinary purposes; the difference between its original and its present size is a factor of safety that has been almost wholly lost.

We may conclude that, with few exceptions, the character of tuberculosis among cattle is that of an effectually concealed disease, the detection of which before it is well advanced and has done great harm is practically impossible through the agency of our unaided powers of observation.

15. A COW AFFECTED WITH ADVANCED TUBERCULOSIS.

The disease is partly located, as is shown by the position of her head, in the lymph glands of her throat. The glands are so much enlarged that, through pressure on the larynx, they greatly narrow the orifice through which air reaches the lung, and yet the visible bodily condition of the cow has remained very good, and is better than that of most dairy cows.

Fortunately we have in the substance known as "tuberculin" an excellent agent for detecting tuberculosis in cattle when all other means of diagnosis fail.

THE MANNER IN WHICH TUBERCLE BACILLI ARE EXPELLED BY TUBERCULOUS CATTLE.

Examinations made at the experiment station of the United States Bureau of Animal Industry showed that tuberculous cows expel tubercle bacilli more commonly with their feces than in other ways.[a] They also expel them with the material drooled and slobbered from their mouths during feeding and ruminating; with the particles of fluid sprayed from their mouths and noses during accelerated expiratory acts; and directly with their milk when their udders are affected. Some authorities assert that tubercle bacilli are expelled directly with the milk when the udder is free from disease, but the observations of the experiment station indicate that this does not occur unless cows are affected with otherwise generalized, advanced tuberculosis.

Milk infected directly through the udder is exceedingly dangerous, because the tubercle bacilli it contains are numerous and of the freshest and most virulent kind. Prof. V. A. Moore, of Cornell University, says:

> It has been shown from all examinations that have been reported of milk from tuberculous cows that about 15 per cent of them give off tubercle bacilli with their milk during the course of the disease. The udders show tuberculosis in about 2 per cent of the cases.[b]

The frequency with which the udders of tuberculous cows are affected is difficult to determine, as is very nicely shown by the observations of the experiment station. For a period of about ten years only 1 per cent of the tuberculous cattle examined were found to have tuberculous udders; while, on the other hand, among the tuberculous cattle examined during the last three to four years fully 6 per cent were found with tuberculous udders. The percentage given by Professor Moore is no doubt very near the truth.

The number of tubercle bacilli expelled from the mouths and noses of tuberculous cattle is probably not as great as the number expelled under parallel conditions of disease from the mouths and noses of tuberculous persons, because cattle cough less frequently and less violently and do not spit. The tubercle bacilli that are expelled with the feces per rectum have their origin in the lung more commonly than elsewhere. They are raised into the mouth and swallowed and on their way through the intestinal tract become evenly mixed with the material that is ejected as feces from the bowels. The result is that the large amount of feces passed by cows, about 30 pounds per day by a cow of average size, introduces an enormous amount of infectious material into their environment when they are affected with tuberculosis, much more than can be safely and economically disposed of so as to make this environment a proper place for the exposure of human food.

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[a] Bureau of Animal Industry Bulletin No. 99.

[b] Cornell University, Bulletin 250, January, 1908.

As the discovery of virulent tubercle bacilli in the feces of tuberculous cattle is of comparatively recent date, and as some harmless bacilli closely resembling tubercle bacilli in appearance are of common occurrence in and about stables and are supposed to be of common occurrence in the feces of cattle, it is desirable to outline briefly the evidence on which the occurrence of virulent tubercle bacilli in the feces of tuberculous cattle rests.

First. The microscopic examinations of the feces of a cow that was being fed small amounts of tubercle bacilli, in the form of cultures added to her drinking water, revealed germs precisely like tubercle bacilli. The cultures of tubercle bacilli fed to the cow were of a virulence too low to cause tuberculosis in cattle. The test of the bacilli in the feces showed that they were virulent for guinea pigs. This experiment proved that tubercle bacilli can pass through the entire length of a cow's intestinal tract and out with her feces without losing their pathogenic virulence.

Second. Numerous microscopic examinations made with the feces of tuberculous cows and with the feces of healthy cows, stabled, fed, and generally kept under precisely the same conditions, revealed that the feces of the tuberculous cows contained bacilli like tubercle germs, and that the feces of the healthy cows did not contain such bacilli. With the exception of a few cases, the tubercle bacilli were not a constant factor in the feces of the tuberculous cows; their occurrence varied from cases in which they were found with every examination to cases in which they were found with daily examinations only once every two to three weeks. This intermittent character of the expulsion of tubercle bacilli in the feces is precisely what should be expected, when we bear in mind that the bacilli have their origin in the lung; that tuberculosis in the lungs of cattle, because of the abundant interlobular connective tissue, is not accompanied by free cavity formation, and that cattle do not cough as freely or as violently as persons affected with lung tuberculosis.

Third. Guinea pigs inoculated with small masses of fresh feces from tuberculous cows that were passing bacilli like tubercle germs per rectum became affected with typical, generalized, fatal tuberculosis.

Fourth. Cultures made from the bodies of guinea pigs that succumbed to tuberculosis induced by the inoculation of fresh feces from tuberculous cows were found to be pure cultures of tubercle

16. HOGS ROOTING IN A MANURE PILE IN A HOG YARD ADJACENT TO A COW STABLE.

The stable contains a herd of tuberculous cattle; more than half of the hogs confined in the yard contract tuberculosis within six months.

17. THREE TUBERCULOUS COWS.

The two on the right of the picture expel tubercle bacilli per rectum with their feces, and probably also with the material slobbered from their mouths during eating. Tubercle bacilli that are passed from the bowels of cows usually have their origin in the lung and throat, from which regions they are coughed into the mouth and swallowed. The visible condition of the cows shows nothing of their dangerous, tuberculous character.

18. A TUBERCULOUS BULL, KNOWN TO PASS TUBERCLE BACILLI PER RECTUM WITH HIS FECES.

When tubercle bacilli can be detected in the feces they are probably numerous, because the opaque character of the material and the facts that the bacilli are isolated from each other and evenly distributed throughout the entire mass make it difficult to find them.

19. AN EXCEPTIONALLY DANGEROUS TUBERCULOUS COW.

At the time her picture was taken she was daily expelling a large number of tubercle bacilli per rectum with her feces Her general condition is good and she shows no symptoms of tuberculosis. Without the tuberculin test she would not have been known to be tuberculous and without other tests her uncommonly dangerous character would not have been suspected. It is not always possible to determine precisely how tubercle bacilli are expelled by individual tuberculous cattle; the tests for this purpose require too much time and careful observation for practical application. It is well to assume that every tuberculous cow expels tubercle bacilli, because, if she does not do so at one time, she will do so sooner or later in the course of the disease.

20. A DANGEROUSLY TUBERCULOUS COW.

Of this cow it is known that she began to expel tubercle bacilli per rectum with her feces eighteen months before her picture was taken. Her condition is that of a fat beef animal. The enormous tuberculous masses sometimes found on post-mortem examination in the bodies of cows like the subject of this picture cause great surprise, and demonstrate that life and seeming health can be maintained under extremely adverse conditions that are of slow and gradual development like tuberculosis or consumption.

21. A DANGEROUSLY TUBERCULOUS COW.

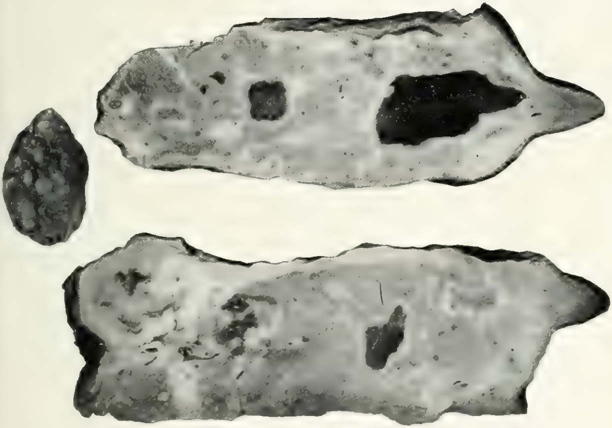
In appearance the subject of this picture is that of a well-kept family cow. She is dangerously tuberculous, because she expels tubercle bacilli from her body per rectum with her feces.

22. A DANGEROUSLY TUBERCULOUS COW, KNOWN TO EXPEL TUBERCLE BACILLI PER RECTUM WITH HER FECES.

Tuberculosis is an infectious disease, and we must always bear in mind that it can be communicated from animal to animal and from animals to persons.

23. AN EXCEPTIONALLY DANGEROUS TUBERCULOUS COW.

Directly after this cow was removed from a dairy herd, because she reacted with tuberculin and not because she showed symptoms of tuberculosis, a small nodule about the size of a pea was discovered under the skin of her udder. Examination of the milk from the quarter of the udder in which the nodule was located revealed the presence of numerous, virulent tubercle bacilli. The cow was permitted to live some time, because it was desirable to use her infected milk for special investigations. When she was killed her udder was found to be in the condition shown in the next picture.

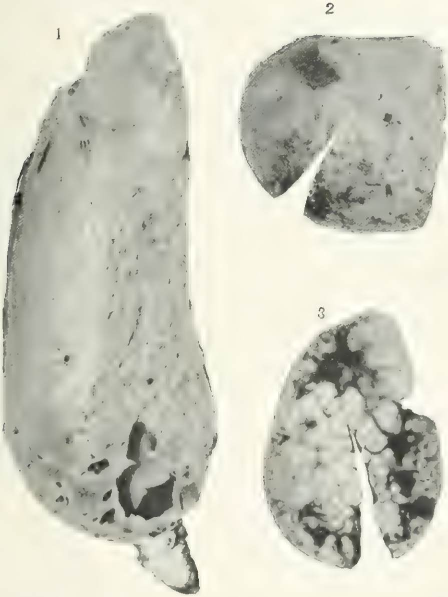


24. SECTIONS OF THE TUBERCULOUS UDDER AND PUBIC LYMPH GLAND OF THE COW SHOWN IN ILLUSTRATION NO. 23.

The cow, in addition to secreting highly virulent, tuberculous milk, also expelled tubercle bacilli from her mouth and per rectum.

25. A DANGEROUSLY TUBERCULOUS COW.

The subject of the picture was kept under observation about two years. She expelled tubercle bacilli per rectum, but her milk remained free from infection until about two weeks before she was killed. Her udder suddenly increased enormously in size and the material obtained from it was found to contain numerous tubercle bacilli. This cow is a rare illustration of the fact that acute tuberculosis may develop suddenly in the udder of a tuberculous cow and thus make her milk extremely dangerous. The next picture shows the condition of the udder found on autopsy.



26. SECTIONS OF THE UDDER AND PUBIC LYMPH GLAND OF THE COW SHOWN IN ILLUSTRATION NO. 25.

The numbers denote 1, longitudinal section of udder; 2, transverse section of udder, and 3, section of pubic lymph gland. The udder is affected with a diffuse, very acute tuberculosis. The lymph gland is many times its normal size, and though it shows no well-marked tuberculous lesions, fluid expressed from it was found on microscopic examination to be loaded with tubercle bacilli.

cilli, and such pure cultures were proven to be virulent for cattle. one instance, a cow inoculated subcutaneously with a culture of is kind became affected with rapidly progressive, generalized tuberlosis, which terminated in death after a few months.

Fifth. Hogs fed with the feces of tuberculous cows contracted pical tuberculosis. The feces were collected under conditions which sure that no infectious material was introduced into them that did ot pass from the bowels of the cows.

The expulsion of tubercle bacilli by cattle per rectum with their eces is one of the most important causes of tuberculosis among hogs, s we may judge from the following illustration, which represents a ommon farm scene a herd of hogs in a hog yard adjacent to a cow table. The cow stable contains a herd of tuberculous cattle; the cattle are not permitted to enter the hog yard and the hogs are not pernitted to enter the cow stable or the field in which the cows pasture. More than half the hogs that remain in the hog yard and root in the manure pile contract tuberculosis within six months.

The relative frequency with which tuberculous cows expel tubercle bacilli per rectum has not been accurately determined. Among 12 cows, collected from several dairy herds for use in an investigation in which a number of apparently healthy tuberculous cows were required, five, or 41⅔ per cent, were found to be passing tubercle bacilli, intermittently, per rectum with their feces. Eighteen months later the number had increased to ten, or 83⅓ per cent; that is, it had doubled, though the majority of the cows still retained their apparently good condition and showed no marked symptoms of tuberculosis. The feces of only a small number of cows that had been affected with tuberculosis three years or more have been examined; they were all found to be passing tubercle bacilli per rectum.

THE APPEARANCE OF CATTLE THAT EXPEL TUBERCLE BACILLI.

When we think of animals afflicted with diseases we usually picture them to our minds as showing distinct variations in their appearance and demeanor from what we regard as healthy and normal. Disease and no symptoms is almost a contradiction, and this seeming contradiction and truly paradoxical condition is one of the important facts about tuberculous cattle.

As no description can define the appearance of an animal as well as a photograph, the following illustrations, Nos. 17 to 26, inclusive, made from photographs, are presented to show the frequently excellent, seemingly healthy condition of dangerously tuberculous cattle. It must be added that the cattle represented in the pictures do not cough; they have excellent appetites and no visible and no audible respiratory difficulties; in all respects they act like healthy animals and perfect harmony exists between their healthy appearance and their healthy conduct. It is only when the tuberculin test is applied to them that their tuberculous condition is revealed, and it is only when the substances that are eliminated from their bodies, feces, saliva, milk, etc., are subjected to microscopic and other tests that the proof is obtained of their dangerous character for public health and for the health of other animals.

Cattle like those shown in the illustrations supply the best proof we can obtain of the possibly dangerous character of dairy cows that are not specially proven to be free from tuberculosis by a careful application of the tuberculin test. These cattle are in excellent condition. Most of them are in better bodily condition than can be expected of ideal dairy cows that give large quantities of milk. They show no symptoms of disease and act precisely like normal, healthy animals. A cow like the subject of illustration No. 23 shows that cows with tuberculous udders may remain undetected until the tuberculin test is applied to them, and a cow like the subject of illustration No. 25 proves conclusively that apparently healthy tuberculous cows may, at any time, without previous warning, suffer an acute extension of the disease with which they are affected to their udders, through which their milk becomes so badly infected with tubercle bacilli that its use in a raw state would be suicidal.

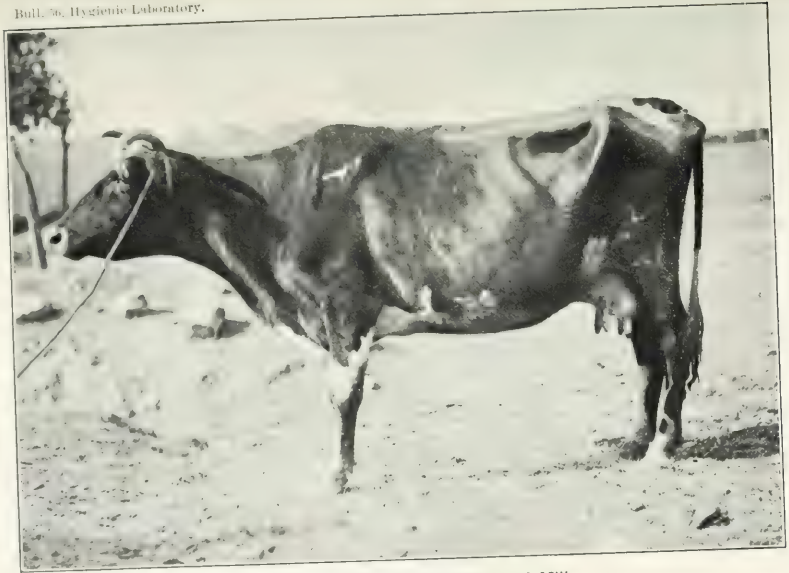
We may conclude that the general appearance of tuberculous cattle, until the disease is near its last stages, or has become generalized, or has dangerously encroached on vitally important organs, is like that of healthy cattle, and that the visibly good or bad condition of cattle is more a question of sufficient feed than of tuberculosis in its earlier stages.

There was a time when veterinarians believed themselves competent to diagnose pulmonary tuberculosis of cattle in its earlier stages by physical examination, but most veterinarians, more particularly those who have had large experience in the examination of bovine lungs by means of ausculation and percussion, are now convinced that this is rarely possible. The broad, flat ribs, the thick hairy hide over the thorax, the transmission of sounds from the intestinal tract, which is enormously large because of the coarse materials ruminants eat, and the common location of tuberculous disease in the dorsal portion of the lung and in the mediastinal space, where it can not be detected at all from without, are a few of the conditions in cattle that interfere with the satisfactory application of the means of diagnosis that are of high value when they are applied to the more tractable and much smaller bodies of persons.

In looking over the pictures given in this article it is desirable that the reader should bear in mind that no special effort was made to obtain photographs from many different sources so as to present ex-

27. A TUBERCULOUS DAIRY COW.

Visibly diseased tuberculous cattle, like the subject of the picture, expel tubercle bacilli almost without exception per rectum with their feces and with the material that is drooled, slobbered, and sprayed from their mouths. It is asserted by some authorities that they also expel tubercle bacilli directly through their udders with the milk. Cows of this kind are altogether too common in dairy herds.



28. A VERY OLD AND VISIBLY TUBERCULOUS COW.

Cows affected with tuberculosis may live, notwithstanding their diseased condition, so many years that their death, when it does come, may be attributed with some justice to the infirmities incident to old age. Two cows, very much like the subject of the picture, were kept under observation for more than six years after it was known that they were affected with tuberculosis. they finally died at an advanced age reached by few cows. This proves conclusively that a tuberculous cow may live many years after she has become a center for the infection of other animals and a menace to public health.

ceptional conditions. All the photographs are those of animals that were among a total of about 50 tuberculous cattle received at the experiment station during the last three years, and among this total of 50 there were at least 25 animals that could well have been used to illustrate the excellent physical condition of dangerously tuberculous cattle and about 40 that could have been used to illustrate simply the healthy, normal appearance of tuberculous cows.

As all persons are not acquainted with the miserable appearance of the cows from which a large part of the public milk supply is derived, and as it will serve as a means to emphasize by comparison the frequently excellent condition of seemingly healthy but, in fact, dangerously tuberculous cattle, two pictures are presented which show by no means the thinnest and most objectionable kind of cows actually found in dairy herds.

HOW TUBERCLE BACILLI EXPELLED BY TUBERCULOUS COWS GET INTO MILK AND DAIRY PRODUCTS.

It has been shown that seemingly healthy, tuberculous cows expel tubercle bacilli directly with their milk and at one end of their bodies with saliva and particles of food and at the other end with feces. Consequently, we may justly conclude, if it can be shown that the material discharged by cattle per mouth and per rectum occurs frequently as a contaminating element in commercial milk, that the milk of tuberculous cows, and also of healthy cows that are stabled and milked together with tuberculous cows, will often be infected with tubercle bacilli.

In one of the publications of the United States Public Health and Marine-Hospital Service the following statement is made about the milk of Washington, D. C., which is no worse than that of other cities:

> In addition to being warm, much of the milk is dirty. Fifty one of 172 samples examined showed no visible deposit in the original container after standing several hours. Fifteen of the samples contained a very small amount of dirt, 98 contained a small amount of dirt, 8 contained much dirt, and 1 contained (mouse?) feces.
>
> The foreign matter (dirt) when examined under the microscope was found to consist of fecal matter, hairs, epithelial and other cells, straw, bacteria, and all manner of extraneous substances that have no place in clean milk.[a]

After several hours' standing in the original containers, 121 of 172 samples, or 70 per cent, of the kind of milk that reaches the consumer showed a visible deposit of dirt, which was found on microscopic examination to be made up in part of fecal matter, or that matter in which tuberculous cows most commonly expel tubercle bacilli.

At the experiment station of the United States Bureau of Animal Industry a large number of samples of milk, purchased from regular city milk dealers under ordinary market conditions, were placed in the tubes of a small centrifuge and rotated fifteen minutes at the rate of 2,000 revolutions per minute. Every sample treated in this way deposited a sediment, and microscopic examinations of the sediment revealed that 98 per cent contained particles of vegetable matter identical in appearance with minute fragments of bovine feces.

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[a] Hygienic Laboratory Bulletin 35, p. 71.

On its face this is a very disgusting condition, but it is unfortunately even more dangerous than its superficial appearance indicates, as the solid impurities that reach the consumer in milk are only a fraction of the total solid impurities with which it has been in contact. Larger masses of feces and other larger, solid impurities that drop into milk or are splashed, sprayed, or otherwise introduced into it from the environment of cows are removed by the straining to which it is subjected before it is poured into the containers in which it is sold; hence only those solid impurities that are small enough to pass through the strainer are found when samples of market milk are examined.

What this means relative to the infection of milk with tubercle bacilli when it is obtained from or in the environment of tuberculous cows is a subject on which it is hardly necessary to enlarge. No firm union exists between the tubercle bacilli and the feces, throughout the entire mass of which they are evenly disseminated. The bacilli are present in an easily detached condition; they are probably washed free from the feces that finds its way into milk, and they are too small to be removed by the strainer. In a test made regarding this matter it was found when guinea pigs were inoculated with normal, fresh milk to which small amounts of fresh feces from tuberculous cows had been added, amounts no greater than commonly fall into the milk pail during ordinary milking, that those inoculated after the milk had been strained succumbed to tuberculosis as rapidly as those inoculated with the milk before it was strained.

That the solid material found in milk as sediment can not be taken as a true measure of the amount of solid, foreign, contaminating material with which it has actually been in contact is shown by the following observation. Among the samples of milk examined relative to fecal content a number were found, with little or no vegetable sediment, that yielded cream in the tubes of a centrifuge discolored precisely like the cream obtained from milk to which uncommonly large quantities of cow feces were intentionally added. Such milk, because of its freedom from sediment and its discolored cream, must be regarded as very dirty and very dangerous milk that has been exceptionally well strained.

The specific gravity of tubercle bacilli is higher than that of milk, and hence it does not seem unreasonable to suppose that they can be removed from it by sedimentation. This supposition would be true milk was a homogenous liquid like water and not an emulsion. Investigations made to determine how tubercle bacilli distribute themselves in milk under different conditions proved that they adhere to the cream globules with a tenacity that can not be broken by a simple difference of specific gravity, even when this difference is reenforced by centrifugalization. The result is, when milk is allowed to stand for cream to rise, or when cream is separated from it rapidly in a centrifuge, the tubercle bacilli, when they are present, rise as abundantly with the cream globules as they gravitate with the sediment and disappear from the intermediate layer or the skim milk, which is practically a homogenous fluid. This holds true when pure cultures of tubercle bacilli are added to milk and also when the bacilli are introduced into it with tuberculous bovine feces, or with pus from tuberculous abscesses, and when they are present because of tuberculous lesions in the udder of the cow from which it was obtained.

These facts almost make it unnecessary to formulate the conclusion that cream, obtained from tuberculous milk, measure for measure, contains more tubercle bacilli than the milk.

Cream is the material from which butter is made, and that butter made from infected cream has the infection transferred to it was proven by repeatedly making butter from infected cream and testing it.

It has been recorded that both strained and unstrained milk to which small masses of feces from cows affected with tuberculosis were added caused tuberculosis in guinea pigs. Cream from such strained and unstrained milk also caused tuberculosis in guinea pigs, and butter made from the cream of such strained and unstrained milk likewise caused tuberculosis in guinea pigs.

Visibly affected tuberculous cows and cows affected with udder tuberculosis are no doubt a serious menace to public health when their milk is used raw in one form or another as human food, but, as dairymen are not exceptionally unscrupulous persons and will rarely sell milk from a visibly diseased cow, and as udder tuberculosis among cows that are not otherwise visibly diseased is rare, we may conclude that the apparently healthy, tuberculous cow, the cow that intermittently expels tubercle bacilli from her body per rectum with her feces, is the most important tuberculous danger for public health that has its origin in the dairy herd. Such apparently harmless, actually dangerous cows not only infect their own milk, but also the milk of the other cows stabled with them, and, as we have seen, such infected milk, both strained and unstrained, equally when it is used as a beverage or as cream or as butter contains live virulent tubercle bacilli.

THE VIRULENCE AND VITALITY OF TUBERCLE BACILLI IN DAIRY PRODUCTS.

Less than ten years ago tubercle bacilli were grouped for all practical purposes in two classes, mammalian and avian, or those which affect man and other mammals and those which affect birds. No one doubted openly that bacilli from cattle, in meat and dairy products, were as injurious for man as those derived from persons. Pulmonary tuberculosis, or consumption of the lungs, was then, as now, the commonest form in which the disease manifested itself, and this was explained by the assumption that the bacilli entered the body more frequently with the breath than in any other way, and that the greatest danger of infection was through dried and pulverized tuberculous material that floated in the air as fine dust.

The beginning of the present century brought a change of views. Attention was called to the fact that the inhalation theory to account for the frequent presence of tuberculosis in the pulmonary tissues had not been proven and that living tubercle bacilli in dust were difficult to find or could not be found at all. The infectiousness of bacilli from animals for man was questioned and the investigation of tuberculosis generally was given a fresh impetus through which many new facts and theories came to light.

As tubercle bacilli in dairy products are mainly derived from bovine sources and enter the body in a moist state, to understand the true significance they have for public health we must give some attention to the infectiousness of tubercle bacilli from bovine sources for man and to the ways in which tubercle bacilli enter the bodies of those who become affected with tuberculosis.

Since Theobald Smith[a] published his studies on different varieties of tubercle bacilli, the evidence in favor of two distinct types virulent for mammals the one found more commonly in bovine and the other in human lesions has grown stronger. But different varieties or types do not necessarily mean different species or even subspecies. As Smith himself stated, "Varieties have been found among nearly all of those specific forms of pathogenic bacteria which have received a considerable amount of attention."[b] The term "varieties" is here clearly used to designate differences of a kind to be expected among the individuals of a large and widespread species, such differences as we know occur among higher organisms than bacteria with a wide geographic distribution. There is a distinct parallelism between a wide geographic distribution of higher plants and animals nd the number and kinds of hosts a pathogenic bacterium may fect; hence, there is no reason why the tubercle bacillus, which has eceived more attention and which affects more species of animals nd more individuals than any other bacterium, should not have been ound to include many different types, the extremes of which would eave us in doubt as to their specific classification if they were not onnected by a chain of transition forms.

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[a] Twelfth and Thirteenth Annual Reports, Bureau of Animal Industry, 1895 and 1896. Jour. of Experimental Medicine, vol. 3, New York, 1898.

[b] Twelfth and Thirteenth Annual Reports, Bureau of Animal Industry, 1895 and 1896, p. 149.

Mohler and Washburn,[a] after a comparison of many tubercle bacilli rom different sources and a careful search of the literature, concluded that the more the subject is studied the more numerous the nstances become in which bacilli of special types are found occurring aturally in animals far removed from the species which may be upposed to be their natural host. They obtained cultures of tubercle acilli from human lesions that were morphologically and biologically bovine types, and in their summary of the investigations of others show that bovine types have frequently been obtained from man and human types from cattle. These investigators,[b] after a prolonged study of the susceptibility of tubercle bacilli to modification, draw the conclusion "that the morphology of tubercle bacilli is their most variable characteristic." They successfully changed the morphology and also the virulence of tubercle bacilli in the course of their investigations and found it possible both to reduce and increase the virulence of tubercle bacilli for different species of animals.

As examples of changes in morphology, the following are instructive as well as interesting: A tubercle culture isolated from sputum was given a more perfect so-called "human" morphological character than it originally possessed by passing it through cats. The same culture was given a perfect so-called "bovine" morphological character by passing it through cattle. A culture isolated from a tuberculous boy was found to be morphologically a bovine type; after fifteen generations on artificial media it was still bovine in character; by passage through cats it became, morphologically, a human type. A culture isolated from bovine tuberculous lesions was found to be morphologically a bovine type; it became morphologically a human type by growth on solidified human blood serum. It is reasonable to assume if human blood serum can effect this change in a morphologically bovine tubercle bacillus from a bovine source that the residence of tubercle bacilli from bovine lesions in the human body may likewise cause a change from so-called "bovine" to so-called "human" morphology.

Mohler and Washburn are not the only investigators who have obtained results to prove that tubercle bacilli may be made to vary in morphology and virulence. In their work they give a history of investigations similar to their own which strengthens the evidence fo the conclusion that we can find nothing in the morphology and virulence of tubercle bacilli to encourage us to undervalue the importanc of those from bovine sources for public health.

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[a] Bureau of Animal Industry Bulletin 96.

[b] Annual Report, Bureau of Animal Industry, 1906, pp. 113-163.

Fibiger and Jensen,[a] who likewise obtained typical bovine bacilli virulent for cattle, from human lesions, recall that the imperial Ger man health office examined 39 cases of primary tuberculosis of th human intestines and mesenteric glands and found that 13 amon them were caused by bacilli of the bovine type. Later investigation made by Fibiger and Jensen are summed up as follows: Though bo vine types of tubercle bacilli are more commonly isolated from bovin lesions and human types from human lesions, there are cultures tha must be considered as transition forms, as they have some of th characteristics of bovine and others of the human type.[b]

Gorter,[c] after a careful study of tubercle bacilli from human an bovine lesions, found 7 among 21 cultures from human sputum whic he regards as identical with the transition forms between human an bovine bacilli which he says are described by Rabinowitsch. He con cludes that human and bovine bacilli are not different varieties, an that the conversion of the one type into the other actually occurs.

Sargo and Suess[d] showed that mutations occur in human tubercl bacilli and other types, which speak against grouping tubercle bacill from animals of different species as special varieties.

Von Behring,[e] who ranks as one of the most widely recognize authorities on tuberculosis, found cultures of tubercle bacilli isolate from man of low virulence for cattle, and others of higher virulenc for them than many cultures of bovine origin. He declares himsel as opposed to the view that bovine tubercle bacilli may be harmles for man, and calls attention to the fact that they generally have higher grade of virulence than human bacilli, and are therefore to b regarded as more dangerous.

The British royal commission on human and animal tuberculosis concluded from its investigations that cow's milk containing bovin tubercle bacilli is clearly a cause of tuberculosis, and of fatal tuber culosis in man, and that a very large portion of tuberculosis con tracted by ingestion is due to tubercle bacilli of bovine origin.

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[a] Berliner Klinische Wochenschrift, Nos. 4 and 5, 1907.

[b] Presented at the joint session of Sections I and VII of the Internationa Congress on Tuberculosis, Washington, D. C., 1908.

[c] Zeitschrift für Tuberkulose, Vol. XI, No. 3, 1907. Also Inter. Centralb. fü d. ges. Tuber. Fors., Vol. II, No. 1, 1907.

[d] Centralb. für Bacteriologie, etc., Vol. XLIII, Part 1, pp. 422-529.

[e] Berliner Tierärz. Wochens., No. 47, 1902.

[f] Jour. Royal Institute of Public Health, Vol. XV, No. 3, 1907.

The nearly unanimous opinion of the members of the recent Interational Congress on Tuberculosis at Washington, D. C., was that he tuberculous dairy cow is a serious menace to public health.

It does not seem necessary to add, as could easily be done, to this vidence to prove that the various existing types of tubercle bacilli are simply mutation forms of one specific organism. The presence of ransition forms between human and bovine types; the occurrence of pure bovine types in human lesions and of human types in bovine esions; the occurrence of bacilli highly virulent for cattle in human esions; the generally greater virulence of bovine types for all species of animals; and the virulence, and greater virulence, of bovine types for anthropoid apes and monkeys,[a] or the animals in the zoological scale most nearly related to man, are all facts that support the conclusion that tubercle bacilli in dairy products are a source of great danger to public health.

It is true that tuberculosis is more commonly an affection of the lung than of other portions of the body. The explanation for this, which was long regarded as satisfactory and is still accepted by many, rests on the assumption that the most important source of tuberculous infection is finely pulverized, tuberculous material, suspended in the air as dust, and the direct exposure of the lung to this dust through the process of respiration. If this so-called "inhalation" theory is true, and as many of those who maintain it assert, tubercle bacilli can not pass through the uninjured wall of the digestive tract and reach organs remote to it without leaving evidences of their passage, then tubercle bacilli in dairy products have no important significance for public health. Therefore to prove that tubercle bacilli in dairy products are dangerous we must give some thought to the mode of infection, or the portal through which the bacilli enter the body.

How strongly the inhalation theory was intrenched in the minds of medical men is well expressed by Aufrecht[b] in the statement that considerable courage was required only a few years ago to characterize it as an unwarranted hypothesis for the wide belief of which no satisfactory evidence had been supplied. He, in 1900, and Baumgarten,[c] in 1901, pointed out that it had not been proven to be the exclusive or even the most important mode of infection with tuberculosis. In 1902 followed the experiments of Nicolas and Descos,[d] confirmed by those of Ravenel[e] in 1903, which proved that tubercle bacilli introduced into the healthy intestinal canal of animals rapidly passed through the uninjured mucosa and appeared in the great thoracic duct on their way to the venous circulation. Nocard and his pupils, Desoubry and Porcher,[a] had earlier shown that the passage of bacteria through the normal intestinal wall and their transference to the blood was possible. Chavreau,[b] in view of the constantly accumulating evidence that pulmonary tuberculosis in man and animals arises from infection through the intestine, calls attention to his investigations from 1868 to 1874, in which pulmonary tuberculosis was brought about by the ingestion of tuberculous material without the production of pathological conditions in the digestive tract.

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[a] Report of the British royal commission in the British Journal, No. 2430, 1907; also, Bureau of Animal Industry Bulletin No. 52, 1905.

[b] Berliner Klinisch Wochens., No. 27, 1907.

[c] Wiener Med. Wochens., Vol. 51, No. 44.

[d] Jour. Phys. et de Path. Gén., Vol. IV, 1902.

[e] Jour. Med. Resea., Vol. X, pp. 460–462.

This earlier work was followed rapidly by other investigations, which proved more and more conclusively that the introduction of tubercle bacilli into the body with food may lead directly to the development of pulmonary tuberculosis, without lesions in the alimentary canal and without intermediate lesions of disease between the digestive and respiratory organs. The most important investigations are probably those of Calmette and his associates, now published in book form.[c]

These investigators claim, and present good evidence in support of their claim, that dust particles never penetrate deeper into the lung than to the first banches of the bronchi; that tuberculosis is constantly a disease of which the infection enters through the intestine; that tubercle bacilli may penetrate the intestinal wall without causing lesions; that the bacilli may pass through the mesenteric glands without causing lesions; that the bacilli frequently cause primary lesions in the mesenteric glands of young experiment animals, but commonly pass through these glands of adult animals and cause primary pulmonary tuberculosis; that tuberculous processes in the lung never begin in the bronchi or alveoli, but constantly in the capillaries, especially in the finest capillary network of the subpleural tissue, etc.

Relative to this localization of the earliest stages of pulmonary tuberculosis, Aufrecht[d] says:

> The initial changes in the apices of the lung, as I have convinced myself by repeated anatomical examinations, do not spread from the terminal branches of the bronchi.

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[a] Comp. Rend. Soc. de Biologie, Vol. XLVII, 1895.

[b] Experiment Station Record, U. S. Dept. of Agriculture, Vol. XIX, No. 2, 1907. (Comp. Rend. Acad. Sci., No. 15, Paris, 1907.)

[c] Recherches expérimentales sur la Tuberculose, effectuées de l'institut Pasteur de Lille, par Calmette et Guérin, P. Vansteenberghe, M. Breton, Grysez, Sonneville et Georges Petit, Paris, 1907. Reviewed in the monthly publication of the International Antituberculosis Association, Tuberculosis, Vol. VI, No. 5, 1907, pp. 256-259; also in Zeitschrift für Tuberkulose, Vol. XI, No. 2, 1907, pp. 163-166.

[d] Berliner Klinische Wochens., No, 27, 1907.

He further says that he has—

> ven the cheesy tubercle in the lung to be associated not with the finer nches of the air tubes, but with the terminal capillaries of the pulmonary eries.

While he is not a special advocate of the intestinal way as the sole de of infection, he ends his article here referred to with these rds: "The inhalation theory for lung tuberculosis is no longer nable." Kohler,[a] who reviews Aufrecht's work, justly remarks that deserves wide recognition, as it supplies important arguments for thorough revision of the older views about the development of lmonary tuberculosis.

Fibiger and Jensen[b] conclude from their own investigations and critical analysis of the reports from numerous widely separated spitals that the former doctrine, which taught that primary intes- nal tuberculosis is a rare disease, can no longer be held as valid. mong 289 children from 1 to 15 years old who had succumbed to rious diseases, 44, or over 15 per cent, were found on autopsy to be fected with primary intestinal tuberculosis. These investigators y that we must, without doubt, return to our former view and re- rd the ingestion of raw milk as an important cause of primary testinal tuberculosis during childhood. This view is in perfect armony with Calmette's experiments, which proved that primary testinal tuberculosis is of commoner occurrence, with infection that ters the body through the alimentary canal, in youth than in adult fe, because tubercle bacilli can pass through the mesenteric glands f adults more readily than through those of children.

Orth[c] makes the statement that even with localized tuberculosis n the lymph glands and the lung we can not exclude the intestine as he portal of entry for the tubercle bacillus. At the international onference on tuberculosis, held in Vienna during September, 1907, he aid that tubercle bacilli can enter the body from the intestinal canal, which might itself, however, remain completely unaffected, but that from the prophylactic point of view the channel of infection was of nly secondary importance, as the object to be aimed at was the destruction of all sources from which infection might take place. As sources of infection he named milk and butter from tuberculous cows nd sputum from tuberculous individuals, and bovine tuberculosis e characterized as undoubtedly infectious for human beings.[d]

Klebs[e] has convinced himself that tuberculosis is a disease of the ymphatic system and may remain such until the end of life, and that infection occurs through the intestines, most frequently with bacilli contained in cow's milk. He claims to have established this as a fact with experiments made at Berne, and published in Virchow's Archives in the early seventies of last century. He says that he has found no reason to change his views, and calls attention to the conclusive manner in which they have been proven by the unimpeachable experiments of Orth, Von Behring, and Calmette.

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[a] Intren. Centralb. für die gesam. Tuber. Forsch., Vol. II, No. 1, 1907.

[b] Berliner Klinische Wochens., Nos. 4 and 5, 1907.

[c] Berliner Klinische Wochens., No. 8, 1907.

[d] Editorial in the New York Medical Record, vol. 72, No. 22, p. 905, 1907.

[e] Deutsch. Medic. Wochens., No. 15, 1907.

Gorter[a] adds his testimony to show that the intestinal mode of infection is not rare, and Bongert[b] showed with rats, as was shown by the experiment station of the Bureau of Animal Industry[c] with hogs and cattle, that the injection of pure cultures of tubercle bacilli into portions of the body as remote as possible to the thorax caused pulmonary tuberculosis without intermediate lesions to connect the location of the disease in the lung with the portal at which the infecting bacilli were introduced.

Baumgarten[d] concluded after experimental studies and a review of the literature that for practical, prophylactic purposes we must consider not only the inhalation theory and ingestion as modes of infection, but all possible ways in which tubercle bacilli may enter the body.

It is not intended to give a complete summary of all the investigations that have supplied evidence to support the fact that tubercle bacilli can and do penetrate the wall of the digestive tract without affecting it and pass to the lung and there cause lesions. It has been amply shown that the intestinal mode of infection for pulmonary and other forms of tuberculosis, unlike the inhalation of tubercle bacilli directly into the lung tissue, is not merely a theory, but a well-established truth, which has forced its way to recognition in the face of considerable opposition. Hence, the frequency with which tuberculosis is a pulmonary disease can not be used as an argument to encourage an undervaluation of tubercle bacilli in dairy products; on the contrary, the mode of infection with tuberculosis, the certainty with which tubercle bacilli may enter one portion of the body and leave it unaffected and cause disease in other portions, condemns dairy products infected with tubercle bacilli as a serious menace to public health.

The relative virulence of tubercle bacilli in moist, opaque substances like milk, cream, butter, and cheese; in dry dust from tuberculous material; in translucent substances like sputum; and in transparent substances like the infectious spray of droplets that escape from the mouths of tuberculous subjects during more or less violent expiratory efforts, also seems to emphasize that the tuberculous cow is a very important source of human tuberculosis.

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[a] Zeitschrift für Tuberkulose, Vol XI, No. 3, 1907; also Intern. Centralb. für die ges. Tuber. Forsch., Vol. II, No. 1, 1907.

[b] Tierärz. Wochens., Vol. XV, No. 29, 1907.

[c] Bureau of Animal Industry Bulletin 93, 1906.

[d] Inter. Centralb. für die ges. Tuber. Forsch., Vol. II, No. 1, 1907.

Cornet[a] is probably the strongest advocate of the dust-inhalation hypothesis. According to his views, dried, pulverized tuberculous sputum is the most important factor for the dissemination of tubercle bacilli and the transmission of tuberculosis from person to person, notwithstanding that he himself calls attention to the rapidity with which the bacilli die upon exposure to light and drying; to the difficulty with which a tough, sticky substance like sputum is pulverized, and to the fact that only a small fraction of a mass of sputum can reach a sufficiently fine state of pulverization to float in the air, or that fine state which he believes necessary for its direct introduction into the finest branches of the bronchial tubes.

Sunlight is the most potent, natural agent for the sterilization of tubercle bacilli; it kills them in less than one hour when they are exposed to its direct rays in translucent layers of infectious pus, and in less than five hours when they are exposed in thick, opaque masses of such pus.[b] Weinzirl[c] asserts that tubercle bacilli, as well as other nonsporulating pathogenic bacteria, are destroyed in from two to ten minutes by direct sunlight, and Koch,[d] Jousett,[e] Flügge,[f] Heymann,[g] Di Donna,[h] Cadéac,[i] and others earlier called attention to the rapidity with which tubercle bacilli are destroyed by desiccation and exposure to light.

If light and drying are the potent factors for the destruction of tubercle bacilli the practical evidence shows them to be, it becomes questionable whether tuberculous sputum, which is so tough that it is difficult to pulverize in a mortar with a pestle after it has been thoroughly dried, ever reaches a state of pulverization in nature that will enable it to float in the air without first wholly losing its infectiousness. Of course, there are scores of ways in which moist tuberculous sputum is dangerous, and hence the rapidity with which light destroys tubercle bacilli and the difficulty with which sputum is pulverized must not be taken as facts that justify or excuse careless spitting.

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[a] Die Tuberkulose, Vienna, 1907, pp. 101-117.

[b] Bureau of Animal Industry Circular No. 127, pp. 17-20.

[c] Dept. Agr. Expt. Sta. Rec., Vol. XIX, No. 3, p. 280, 1907. (Jour. Infect. Diseases, May, sup. 3, pp. 128-153.)

[d] Cornet, Die Tuberkulose, p. 41. Vienna, 1907.

[e] Wiener Med. Wochens, 1901, No. 28, p. 1366

[f] Zeitschrift für Hygiene, vol. 38.

[g] Editorial Jour. Amer. Med. Asso., Oct. 12, 1901.

[h] Centralb. für Bact. und Parisitenk, Vol. XLII, No. 7.

[i] Le Bulletin Médical, Sept. 5, 1906,

The vitality and virulence of tubercle bacilli in dairy products is very different from their rapid destruction in sputum, and we must also bear in mind that they are not on the floor, or in the air, or in other places from which they may or may not gain access to our bodies, but that they are contained within articles of food with which they will certainly be introduced into our bodies.

Broërs,[a] whose work on tuberculous dairy products is based on careful observations, found that tubercle bacilli will live three days in milk, even when it has undergone changes to make it unfit for use as food, twelve days in buttermilk, and that they certainly remain virulent in butter three weeks. As milk and buttermilk are rarely used in a raw state after they are more than three days old, it is not necessary to show that the tubercle bacilli they may contain will remain alive and virulent longer than Broërs has recorded. The length of time the bacilli remain virulent in butter is another matter, and regarding it the available data are very contradictory, as is shown by Cornet,[b] who says:

> Laser could find no live tubercle bacilli in butter after twelve days; Heim records that all tubercle bacilli eventually die in butter, and that their maximum life in it is thirty days; Gasperini found a reduction of virulence after thirty days, though the bacilli were still alive after one hundred and twenty days; and Dawson did not observe a reduction of virulence until after three months, and claims to have produced tuberculosis in a guinea pig by inoculating it with butter eight months old.

As the two extremes, twelve days and eight months, are too far apart to be satisfactory, an investigation relative to this matter was undertaken at the experiment station of the Bureau of Animal Industry.[c]

Butter was made from the milk of a cow affected with udder tuberculosis, and tested from time to time by making guinea-pig inoculations with it. The butter was salted at the rate of 1 ounce of salt to the pound of butter, and the conclusions drawn regarding it are as follows:

> The guinea pig inoculations show that tubercle bacilli in ordinary salted butter undergo no attenuation in forty-nine days; that they are still highly virulent after ninety-nine days, or more than three months, and that they are still alive after one hundred and thirty-three days.

Since these tests were made it was found that the bacilli are still alive after one hundred and sixty days, which indicates that Dawson's period of eight months is not an exaggeration.

As the investigations of the experiment station regarding the long-retained virulence of tubercle bacilli in butter called out a popular riticism to the effect that the inoculation of guinea pigs was not a ufficient test to show that such bacilli are dangerous when they are ngested, the following experiment was made:

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[a] Zeitschrift für Tuberkulose, Vol. X, No. 3.

[b] Die Tuberkulose, Vienna, 1907, p. 121.

[c] Bureau of Animal Industry Circular No. 127.

Four hogs, weighing 125 pounds each, were tested with tuberculin o make sure that they were free from tuberculosis, and then placed n four separate disinfected pens. Each hog was fed 1 ounce of utter daily in addition to its other feed: the butter was of the kind sed for the guinea-pig-inoculation tests: the feeding was continued hirty days. This butter consisted of several different lots, the youngest of which was 90 days or 3 months old when it was fed to he hogs. The amount of butter received daily by each hog was less han the average person of the same weight eats, and the total amount received by each hog was less than 2 pounds.

Several months after the feeding of butter was discontinued the hogs were killed and examined post mortem, and three of the four were found to have contracted tuberculosis.

More direct evidence to prove that tuberculosis is contracted from infected food, and more direct evidence to prove that tubercle bacilli remain alive and virulent a quarter of a year in ordinary butter, would be difficult to obtain.

In oleomargarine tubercle bacilli may also remain alive long periods of time, probably as long as in butter, which it closely resembles in general character. In cheese the germs are especially dangerous when they occur in fresh products, like cottage cheese, but that even those cheeses which require some time to ripen are not wholly safe is shown by the fact that Prof. F. C. Harrison proved that tubercle bacilli may remain alive in Cheddar cheese, a standard American variety, one hundred and four days.[a]

We may conclude, as far as it is possible to test the vitality and virulence of tubercle bacilli from different sources and in different environments, that those from cattle are, as a rule, the most virulent, and that it seems to be clear that dairy products generally, and butter especially, supply an ideal medium for the preservation of both the life and virulence of tubercle bacilli.

THE PROPORTION OF TUBERCULOUS COWS AMONG THOSE IN USE FOR DAIRY PURPOSES.

General statistics from which we can determine the percentage of dairy cows affected with tuberculosis are not obtainable. In the District of Columbia about 17 per cent of the cows tested with tuberculin reacted, and in the State of New York the figure among those tested is about 30 per cent. It does not absolutely follow from this that the cattle of New York State are more commonly tuberculous than those of the District of Columbia, because in both places the number tested is only a small portion of the total number in use, and the percentages of tuberculosis obtained respectively may have been influenced largely by the motive that prompted the application of the test. When tests are made at the request of those who own dairy herds, it may be assumed that the owners of exceptionally good herds will be in the majority, and the percentage of tuberculosis discovered will be low. If, on the other hand, the tests are largely forced for the protection of public health because tuberculosis is suspected among the tested animals, the percentage of tuberculosis found will rise to the maximum figure.

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[a] United States Bureau of Animal Industry, Annual Report, 1902, p. 228

The writer has personally tested a large number of dairy herds in widely separated localities, and in all his tests did not have the good fortune to find a single herd entirely free from tuberculosis. Most of these herds, however, were tested at the request of owners who had some reason to suspect tuberculosis among their cattle, and hence this discouraging experience can not be used as a reason for assuming that few perfectly healthy dairy herds exist.

From the figures and estimates that are available it seems fair to conclude that not less than 20 per cent of our dairy cows are tuberculous, and that tuberculosis occurs to some extent in about 30 per cent of our dairy herds. These are believed to be conservative figures, but they must be taken strictly as having purely and simply the value of an estimate. In some European countries, where better statistics are available than in the United States, it is safe to conclude that not less than 40 per cent of all dairy cows are tuberculous, and this high percentage will be reached among our dairy cattle before long unless vigorous means are used to prevent the further spread of tuberculosis among them.

THE FREQUENCY WITH WHICH DAIRY PRODUCTS HAVE BEEN PROVEN TO CONTAIN TUBERCLE BACILLI.

The truest test of the measure in which the public is exposed to tubercle bacilli from bovine sources is the frequency with which tubercle bacilli occur in dairy products.

Without reviewing investigations of older date or those made in foreign countries, four comparatively recent investigations made in America show how common the occurrence of virulent tubercle bacilli in milk is. The largest of the four investigations showed that 15, or 6.7 per cent, of 223 samples of milk contained tubercle bacilli. The milk was obtained from 102 dairies, among which 11, or 10.7 per cent, were distributing infected milk.[a] The second investigation showed that 2, or 2.7 per cent, of 73 samples of milk ontained tubercle bacilli.[a] The third investigation showed that 2, r 5.5 per cent, of 36 samples of milk contained tubercle bacilli. he milk was obtained from 26 dairies, among which 2, or 7.7 per ent, were distributing infected milk.[b] The fourth investigation howed that 17, or 16 per cent, of 107 samples of milk contained ubercle bacilli, and that among 8 samples of commercially pasteur zed milk one was found that contained live tubercle bacilli.[c]

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[a] J. F. Anderson, United States Public Health and Marine Hospital Service, Hygienic Laboratory Bulletin No. 41, pp. 163-192.

The four investigations taken together show that among the 439 amples of milk 36, or 8.2 per cent, were infected with live, virulent ubercle bacilli.

The fact that one among eight commercially pasteurized samples of nilk contained living tubercle bacilli is conclusive proof that some of he so-called "pasteurization," commercially practiced, is worse than useless, and has the evil tendency to quiet the mind regarding grave dangers it does not correct.

It is a serious charge against the milk commonly sold by dairies to say that fully 1 sample among every 12 contains living, virulent tubercle bacilli, and yet this is the most favorable conclusion we can draw from four of the most recent and most reliable investigations with which the writer is acquainted.

A further analysis of the two among the four milk investigations that give the number of dairies from which milk was tested proves that the conditions are worse than their superficial appearance indicates. These two investigations show that 17, or 6.5 per cent, of 259 samples of milk obtained from 128 dairies were infected, and that the infected milk was sold by 13, or 10 per cent, of the dairies. The two investigations also show that the total number of samples of milk obtained from the 13 infected dairies is 31, of which 17 were infected and 14 were free from infection. Hence, the difference between the percentage of infected milk samples and the percentage of infected dairies can not be explained on the assumption that it is due to the more frequent duplication of tests with milk from the noninfected than from the infected dairies; it is shown on the face of the evidence that the difference of the two percentages is due to the fact that infected dairies distribute infected milk intermittently and not continuously.

The intermittent distribution of infected milk by infected dairies is not only interesting because it may be related to the intermittent expulsion of tubercle bacilli by cattle with their feces, but also because it justifies that we should draw the conclusion from the milk tests we have under consideration that a larger proportion of dairies than even 10 per cent must be classed as infected.

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[a] J. R. Mohler, same work as above, pp. 493-495.

[b] Unpublished work of the experiment station of the United States Bureau of Animal Industry.

[c] Dr. Alfred Hess, of New York. Paper presented at the International Congress on Tuberculosis, Washington, D. C., 1908.

To obtain further information regarding the intermittent distribution of tuberculous milk by infected dairies,[a] samples of milk were bought from a dairy, from which several months previously a sample of milk had been obtained that was found to be infected with virulent tubercle bacilli, on 30 different days and injected into guinea pigs. Among the 30 samples the second, third, and eighth were found to contain tubercle bacilli; the remaining 27 were not infected. If we add the sample of milk which first showed the infected character of the dairy to the 30 later samples, we have 31 from one source among which 4, or about 13 per cent, were found to contain tubercle bacilli. It does not require much reasoning to conclude from this evidence that the chances for discovering an infected dairy by testing one sample of milk from it may be equal to only 13 per cent, and the chances that the one test will not reveal the infected character of a dairy may be nearly eight times as great as the chances that it will.

I do not wish to create an exaggerated idea of the proportion of dairies that intermittently distribute tubercle bacilli in milk, because the facts are so grave that, without exaggeration, they are almost beyond belief. It is well, however, to know the truth, and through knowing it, to be convinced that the milk of no dairy can be accepted as permanently free from tubercle bacilli unless it is obtained in a clean, wholesome environment from cows shown by the application of the tuberculin test to be free from tuberculosis.

The available data regarding the frequency with which tubercle bacilli occur in butter and other dairy products than milk are very meager for the United States, but when we know that tubercle bacilli in milk are transferred to the cream, butter, cheese, etc., made from it, we can readily infer how commonly these products are infected. Relative to the infection of cream and butter the following paragraph from a report of the United States Secretary of Agriculture is very significant:[b]

> The examination of sediment taken from cream separators of public creameries throughout the country has demonstrated the presence of tubercle bacilli in about one-fourth of the samples.

In a recent publication of the United States Bureau of Animal Industry it was pointed out that both the tendency of tubercle bacilli to rise with cream and a comparison of European statistics relative to the frequency with which tubercle bacilli have been detected, respectively, in milk and butter indicate that when tubercle bacilli are present in milk they will no doubt be present in greater concentration in cream and butter.[a]

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[a] Unpublished work of the experiment station of the United States Bureau of Animal Industry.

[b] Annual Report of the Secretary of Agriculture, Washington, D. C., 1907, p. 30.

We can protect ourselves against the tubercle bacilli that are distributed in milk by practicing home pasteurization; but with butter this is not possible; and it is therefore desirable that the milk or cream used in the manufacture of butter should either be obtained from cows certainly free from tuberculosis, or that it should be pasteurized before it is used.

SUMMARY.

We have seen that tuberculosis is the commonest disease of both persons and dairy cows, and that persons and dairy cows are its commonest victims; we know that dairy products are indispensable, and that they are more commonly eaten in a raw state than other products from animals; we have seen that tuberculosis is an insidious, chronic disease, and that tuberculous cows often expel tubercle bacilli long before they show signs of their diseased condition; we have seen that milk is almost invariably contaminated with the material in which tuberculous cows most commonly expel tubercle bacilli from their bodies;[b] we have seen that milk is so often infected with virulent tubercle bacilli that unless we know it to be derived from cows that are certainly free from tuberculosis it is not safe to use it in a raw state; we have seen that tubercle bacilli in milk are transferred to the cream, butter, and cheese made from it, and may occur in these products in greater concentration than they had in the milk from which they were derived; we have seen that no better medium for the preservation of the life and virulence of tubercle bacilli can be found than the moist, bland, and opaque character of butter offers; we have been told that the medical profession is well-nigh unanimous in the view that tubercle bacilli from the bovine source in dairy products are a serious menace to public health; and we have seen that, in our fight for the suppression and eventual eradication of tuberculosis, we must seek to make harmless all the sources from which tubercle bacilli are expelled. Add to this that the available evidence regarding different types of tubercle bacilli shows that bovine types have been found in human lesions and human types in bovine lesions; that transition forms connect bovine types directly with human types; that the most variable feature about a tubercle bacillus is the character that is used to classify it as a special type; that tubercle bacilli of human types have been converted into bovine types and those of bovine types into human types, and that tubercle bacilli of the so-called "bovine" type are, as a general rule, more virulent than those of the human type for all animals, including manlike apes, and the conclusion is almost forced upon us that the tuberculous dairy cow is, to say the very least, one of the most important sources of tubercle bacilli with which we have to deal.

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[a] Bureau of Animal Industry Circular 127, 1908, pp. 4-5.

[b] The expulsion of virulent tubercle bacilli with the feces from the bodies of apparently healthy, tuberculous cows has been confirmed since this article was written by the British Royal Commission on Human and Animal Tuberculosis. See Third Interim Report, London, 1909.

The commoner occurrence of tuberculosis in the lung than in other parts of the body should not encourage us to undervalue tubercle bacilli concealed in articles of food, as it has been shown that infection may penetrate to the lung as easily by the way of the intestine as directly through the trachea and bronchi; in fact, a critical consideration of the two modes of infection, inhalation and ingestion, shows that the latter is in better harmony with known facts than the former.

As there is a lack of clearness about the popular conception of the channels through which disease germs may penerate into and injure the lung, it may be well to devote a few additional paragraphs to this subject.

The inhalation of infectious material directly into the lung requires, first, that the infectious material must be suspended in the air, and, second, that the infectious material must remain in suspension while the air passes through a long, narrow, tortuous, moist-walled system of channels. We have seen how difficult it is to pulverize the tuberculous material, sputum, from which fine tuberculous dust is supposed to arise, and we have seen how rapidly tubercle bacilli are destroyed when they are exposed to light and drying; hence we may conclude that dust charged with live, virulent tubercle bacilli is by no means plentiful. But even if tuberculous dust was abundantly suspended in the air, its penetration into the finer bronchial tubes of the upper portions of the lung, where tuberculous processes most commonly begin, would necessitate a suspension of the laws that govern the relative movements of substances of higher and lower specific gravity actuated by the same force.

When a moving fluid holds solid particles of relatively higher specific gravity in suspension, every change in the direction of the movement will cause the heavier, solid particles to move somewhat more tangentially than the lighter fluid. If the movement occurs in a tube, the heavier particles will be thrown with more or less force against the wall of the tube. When the heavier particles are a dry dust and the fluid is a dry gas like air and the movement occurs in moist walled channels like the air tubes, the dust will be thrown against and adhere to the moist walls, and the air will be thoroughly purified long before the number of turns or changes of direction have been made that occur in the air passages from the exterior of the body to the finer bronchial tubes. Now, the larger air tubes on which dust may be deposited are covered with a ciliated epithelium or cells, which have fine hairlike processes that are in constant motion, and the motion is of a kind that tends to move dust, etc., outward and not farther into the lung.

From this it should readily be seen that the inhalation theory to account for the infection of the lung is simple only when we fail to analyze it, and that analysis shows it to be a practically impossible hypothesis.

The normal channel through which material from without enters the body is the digestive canal. It has been shown by Nicolas and Descos, Ravenel, Schloszmann and Engle, Calmette and his associates, and other bacteriologists and pathologists too numerous to mention, that tubercle bacilli may penetrate rapidly through the healthy walls of the intestines and reach the great thoracic lymph duct. The thoracic lymph duct empties its contents into one of the large veins that communicate with the heart; mixed with the blood in this vein the material from the duct enters the heart and is pumped directly to the lung, where it is filtered through the lung capillaries, which are the finest and most complex capillaries of the body. If we recall that the careful anatomical examinations made by Aufrecht and by Calmette and his associates proved that the tuberculous processes in the lungs have their beginning in the finer capillaries and not in the finer air tubes, we are in a position to conclude that infected food much more than infected air is to be dreaded as a cause of tuberculosis.

Tuberculosis among dairy cows is so common and wide spread that we can not hope to clean all dairy herds of the disease for some time to come; hence it is necessary for the protection of health to avail ourselves of the one expedient which is immediately at hand, and that is pasteurization; and pasteurization should not be restricted to milk, but all milk, cream, etc., used in the manufacture of butter, cheese, and other dairy products, should be pasteurized, unless it is obtained from healthy, nontuberculous cows that are stabled under hygienic conditions in an environment wholly free from tuberculous infection.

The elimination of tuberculosis from the dairy herd is urgently recommended, not only because the protection of public health requires it, but also because tuberculosis among cattle is a serious cause of pecuniary loss, so serious indeed, that, from the strictly economic point of view, it must be regarded as the most important problem those who are interested in animal husbandry can undertake to solve.

16. SANITARY INSPECTION AND ITS BEARING ON CLEAN MILK.

SANITARY INSPECTION AND ITS BEARING ON CLEAN MILK.

By Ed. H. Webster,
Chief of Dairy Division, Bureau of Animal Industry, Department of Agriculture.

In discussing this subject it will be assumed that the herd is in perfect condition as regards health, that there are no persons employed in or about the dairy suffering from any communicable disease, and that the water supply has been examined and found pure. This assumption is made with the understanding that if any of these conditions are not complied with the milk will be debarred from the market, or under certain prescribed regulations be allowed sale after pasteurization.

CLEAN MILK.

It is evident that in nature's scheme for the nourishment of the young milk was never intended to see the light of day, and if suckled from a normal, healthy gland is the perfect food for the offspring. In this natural method of nourishment there is little possibility of contamination from outside sources. As soon as the artificial method of drawing milk is resorted to there enters a whole set of conditions entirely new and different. The milk then comes in contact with the air, the vessel into which it is drawn, and with particles of dirt from many sources.

The problem of securing clean milk—i. e., milk as near as possible to the condition as it exists in the udder—is the problem of dairy sanitation. To put it in another way, it is the problem of reducing contamination from all outside sources to the least possible factor.

WHAT IS CONTAMINATION.

If the mere presence of solid particles of dirt so frequently found in the milk were the only damage wrought, the question would resolve itself into the simple operation of straining or passing the milk through a clarifier. The presence of solid dirt is, however, an indication of much more serious conditions. Bacteriology teaches that every particle of dirt, whether it seems to the eye a source of contamination or not, carries with it great numbers of bacteria, and that milk at ordinary temperatures, 65° F. to 100° F., is an excellent medium for their growth, and most of the changes that take place in milk can be traced directly to such action.

Neither straining nor clarifying will remove the bacteria from the milk, hence the necessity of keeping the dirt out, not straining it out.

SOURCES OF CONTAMINATION.

From the act of milking to the final consumption of the milk the possibilities of contamination are many and varied in character. Everything that comes in direct contact with the milk may be a source of trouble, and many things may act indirectly and seriously affect the results desired.

MILKING.

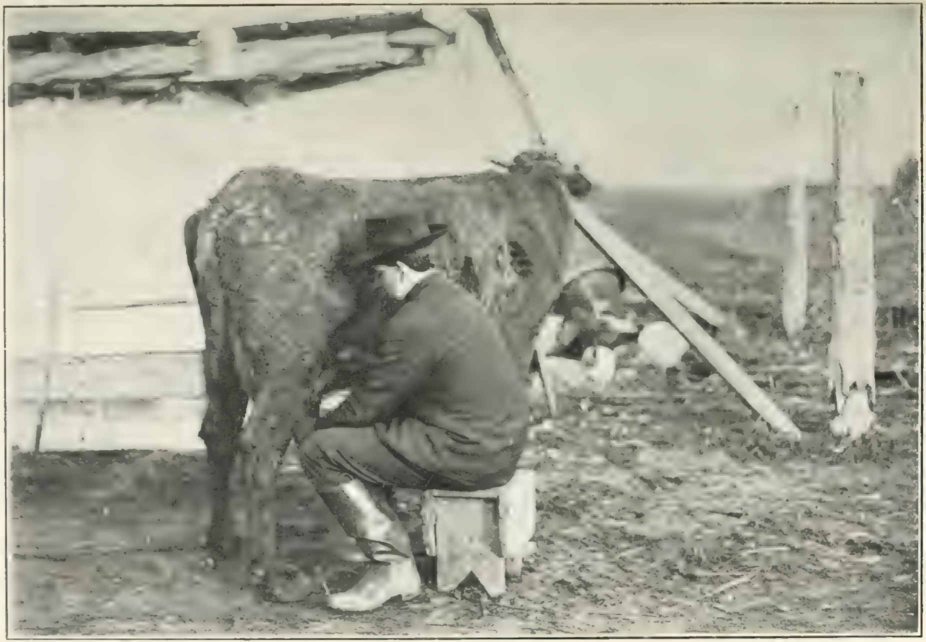
The first contamination usually begins with the act of milking. If the udder and flanks of the cow are covered with the dirt of the yard or stable the process of milking will dislodge a greater or less portion of this filth, causing it to fall into the pail.

The amount of filth that may be on a cow will depend very much on the condition of the stable yard, and floor, gutter, and bedding in the stables.

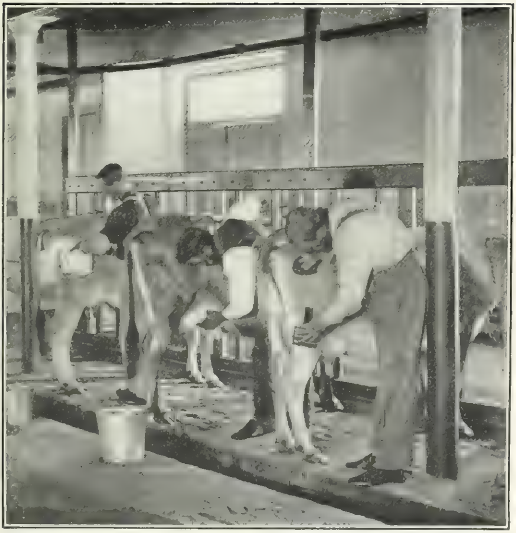
The following illustrations are used to better show conditions which are too common the country over. The condition of the cow shown in Fig. 29 is not exaggerated. The milker is probably all unconscious that he is sowing the seeds of contamination and destruction, which may sooner or later cause the death of infants who are unfortunate enough to be fed from milk produced under such circumstances. If he is aware of this fact he is criminal in purpose and intent, and the most stringent penalties should be provided to stop such work. Figs. 31, 32, 33, and 34 show exteriors and interiors of barns which will contribute to such a fearful condition.

As to the amount of filth that will get into the milk and the result on the product, reports from the Illinois Experiment Station and Storrs (Conn.) Experiment Station are here quoted:

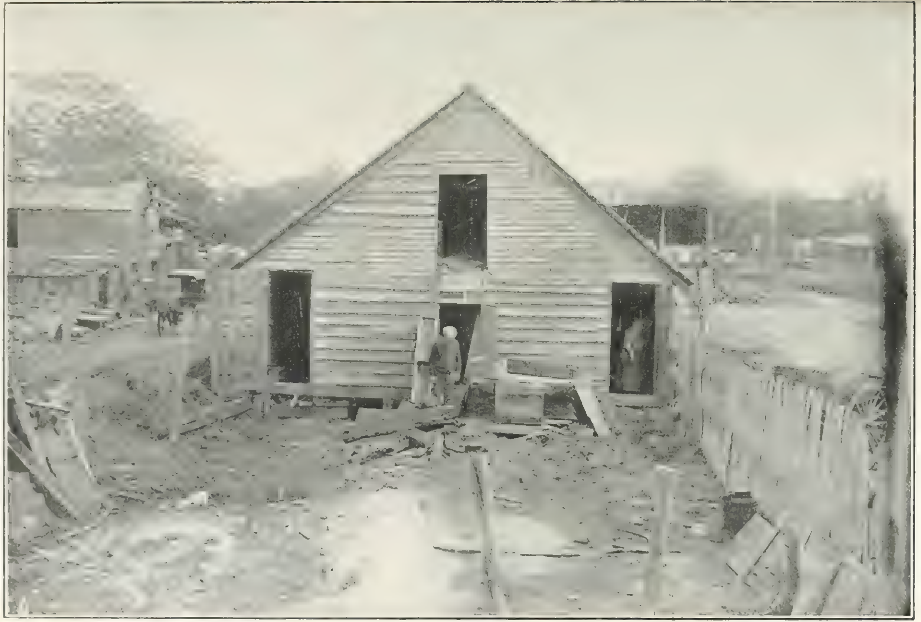
> The average weight of dirt which falls from muddy udders during milking is 90 times as great as that which falls from the same udders after washing, and when udders are slightly soiled it is 32 times as great. (Bulletin No. 84, Illinois Experiment Station.)
>
> Wiping the flank and udder of the cow with a damp cloth just before milking is a very efficient method for reducing the number of bacteria which falls into the milk pail. (Stocking. Bulletin No. 42, Storrs Experiment Station.)



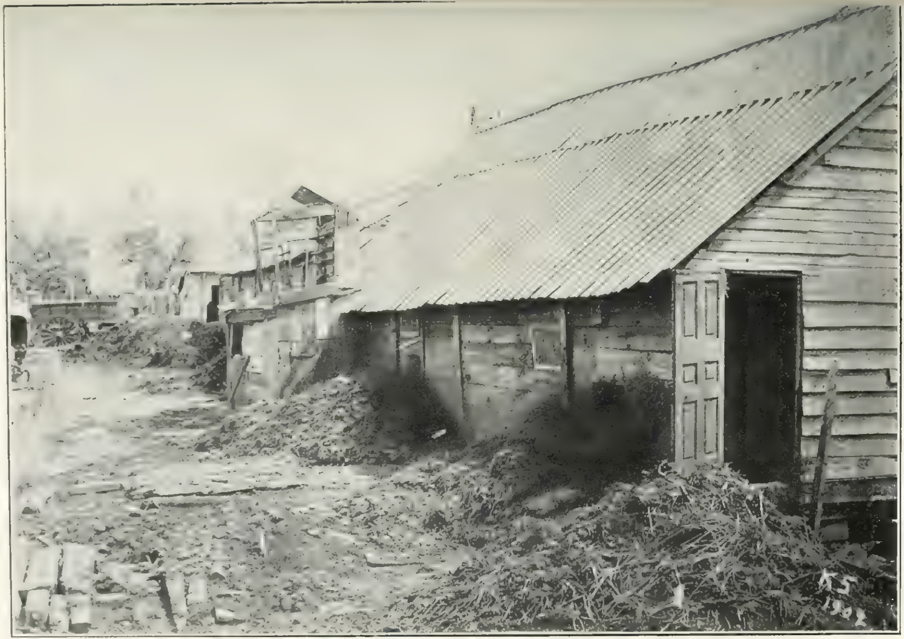
[illegible] DIRTY FLANKS. A COMMON CONDITION IN WINTER. FLANKS BECOME CAKED WITH MANURE, WHICH THERE IS OFTEN NO THOUGHT OF REMOVING. THIS IS THE SOURCE OF MOST OF THE DIRT FOUND IN MILK IN WINTER TIME.



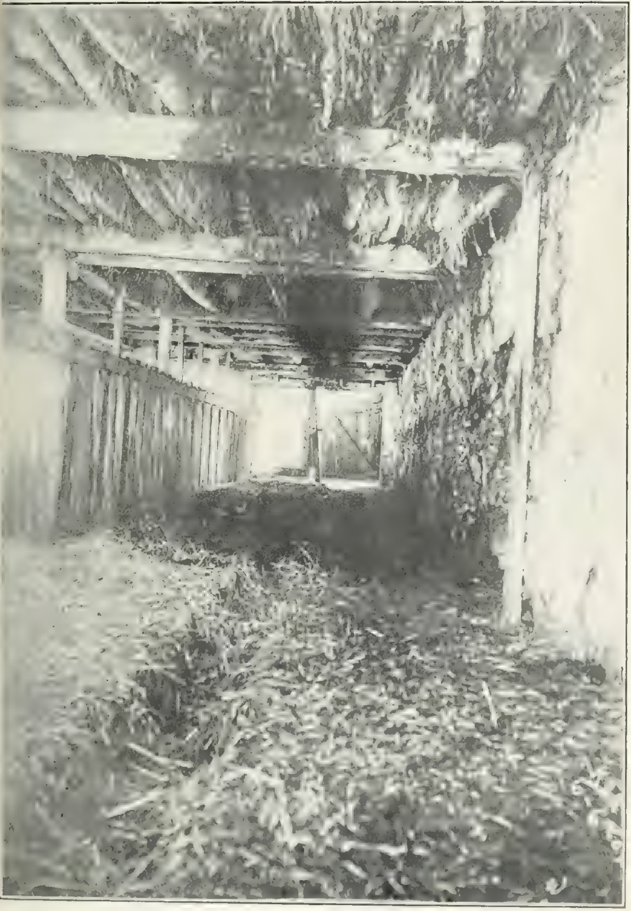
30. CLEANING COWS PREPARATORY TO MILKING. A SIMPLE OPERATION REQUIRING NO OTHER OUTLAY THAN A LITTLE TIME.



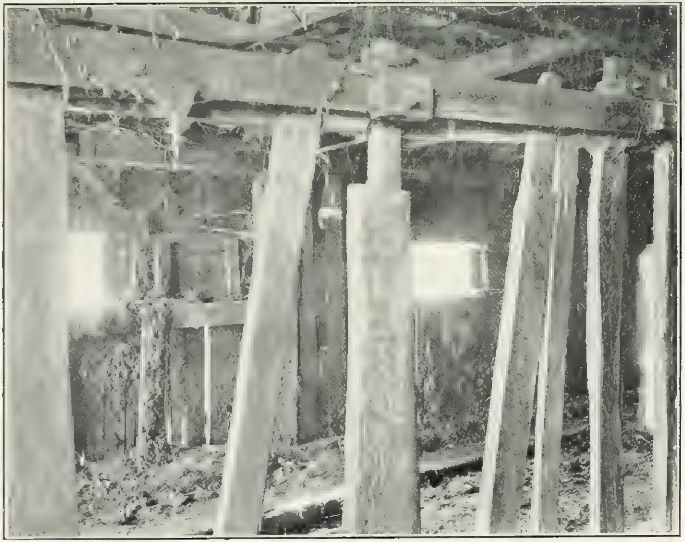
[illegible] STABLE YARDS OF THIS TYPE ARE ALL TOO COMMON. THE COWS ARE COMPELLED TO WADE KNEE DEEP IN MANURE IN ORDER TO GET INTO THE STABLE. MUCH OF THE FILTH ON LEGS AND TAIL FROM THIS SOURCE GETS INTO THE MILK.



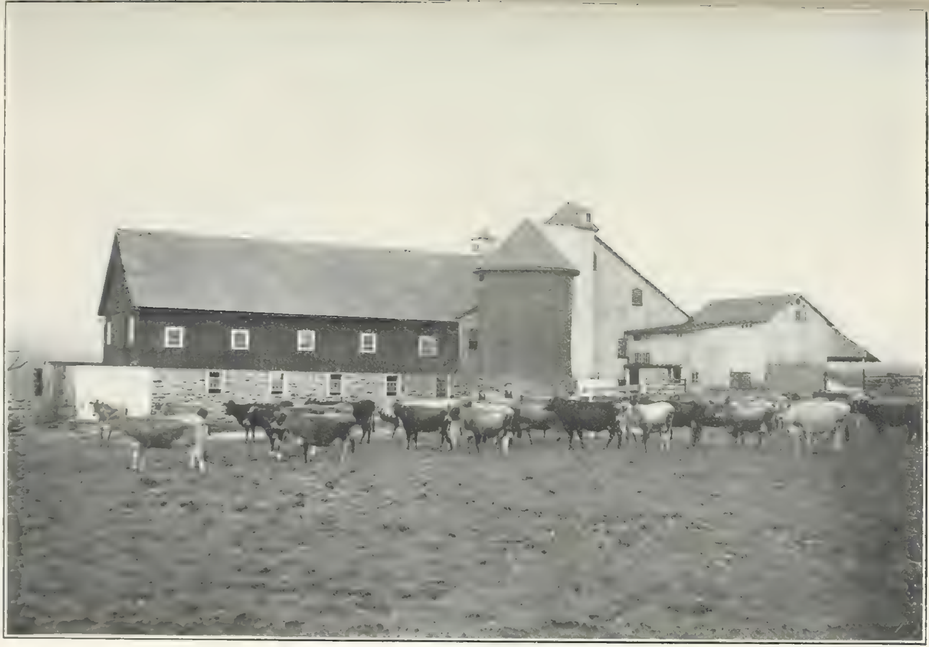
32. ILLUSTRATES THE SAME CONDITIONS AS 31.



33. FILTHY WALLS, FLOORS, AND CEILINGS. A CONDITION FREQUENTLY MET WITH IN OLD BARNS. CEILING FULL OF COBWEBS AND DUST, WALLS AND FLOORS SHOW LITTLE EVIDENCE OF CLEANING. CLEAN MILK CAN NOT BE PRODUCED IN SUCH A PLACE. "THIS IS THE KITCHEN WHERE BABY'S BREAKFAST IS PREPARED."—DR. SANTEE.

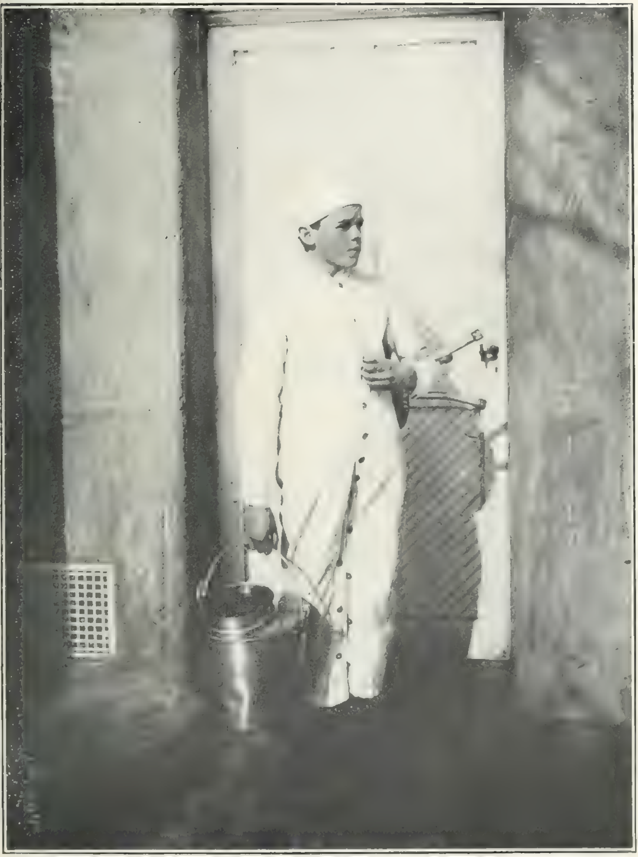


34. ILLUSTRATES THE SAME CONDITIONS AS FIG. 33.

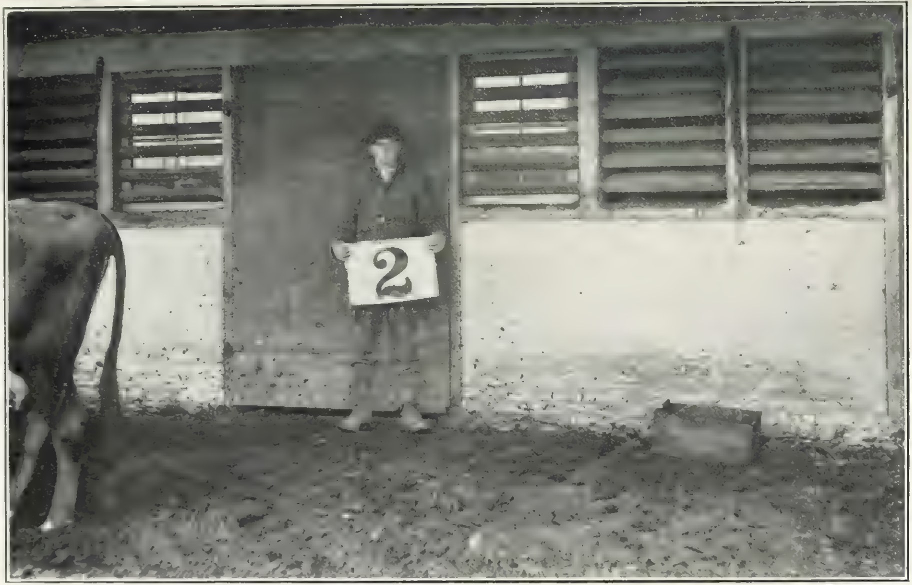


5. SHOWS A CLEAN BARNYARD AND WELL-LIGHTED BARN.

36. A CLEAN LIGHT, AIRY INTERIOR. MILKERS AT WORK ARE DRESSED IN CLEAN WHITE SUITS AND CAPS. COWS ARE CLEAN. AN IDEAL PLACE.

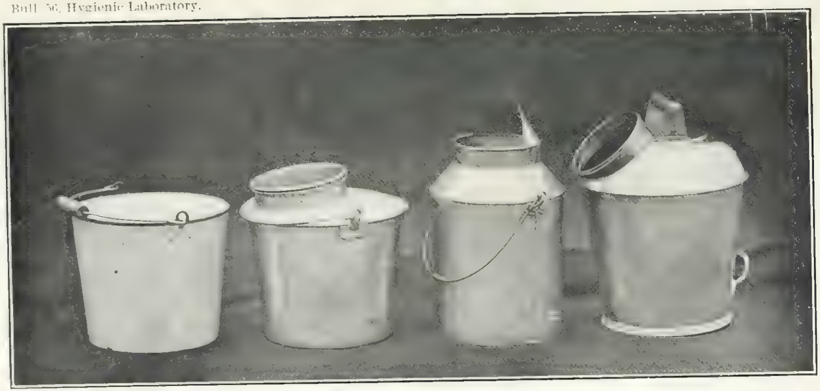


37. GOOD TYPE OF MILKING SUIT AND PAIL

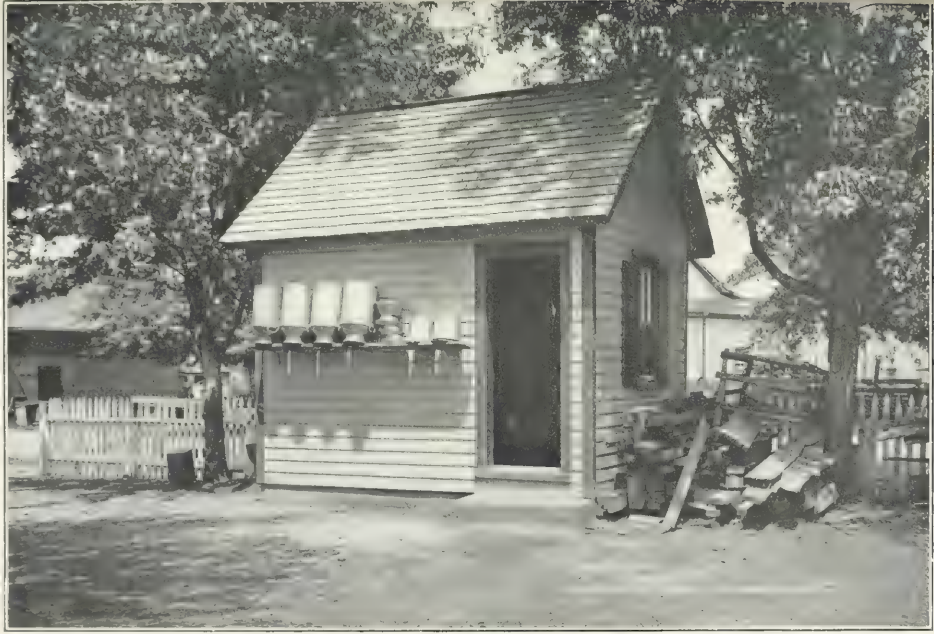


[illegible] A BLIND COMPLIANCE WITH REGULATIONS CONCERNING GLASS WINDOWS IN BARN. OWNER EVIDENTLY HAD NO CONCEPTION OF PURPOSE OF WINDOWS.

9. FOLLOWING THE LETTER BUT NOT THE SPIRIT OF A LAW WHICH DID NOT ALLOW MILK TO BE KEPT IN STABLES AFTER MILKING, BUT FAILED TO MENTION THE HOG HOUSE.

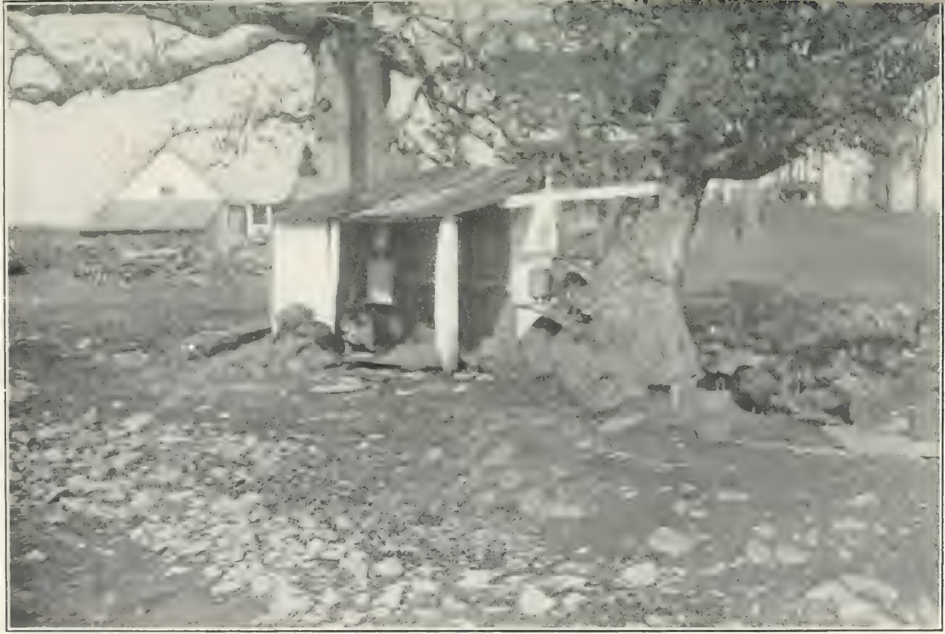


40. TYPES OF MILK PAILS. NARROW-TOP PAILS ARE BEST.



41. A GOOD TYPE OF INEXPENSIVE MILK HOUSE.

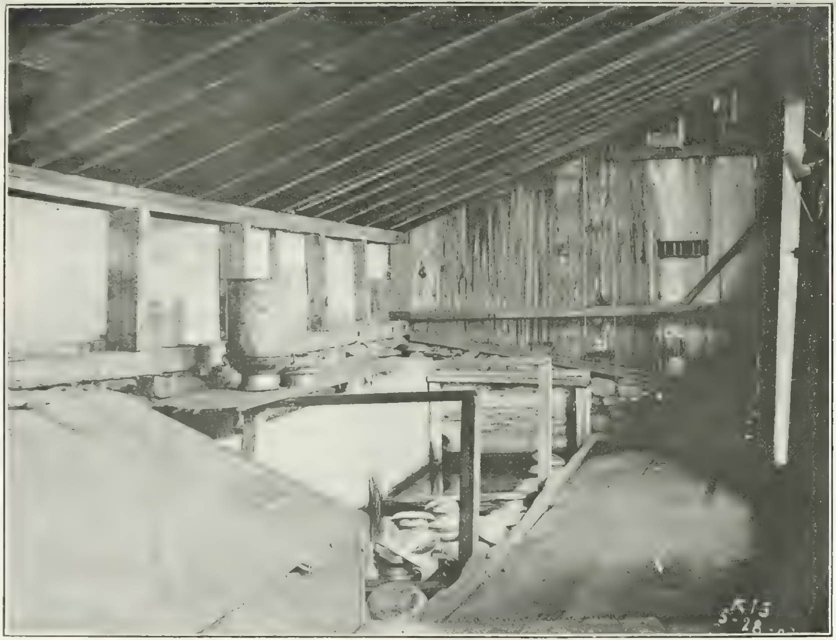
42. THE INTERIOR OF FIG. 41. CLEAN AND NEAT.



4. A MERE PRETENSE OF A MILK HOUSE. TURKEYS ROOSTING AROUND THE MILK UTENSILS.

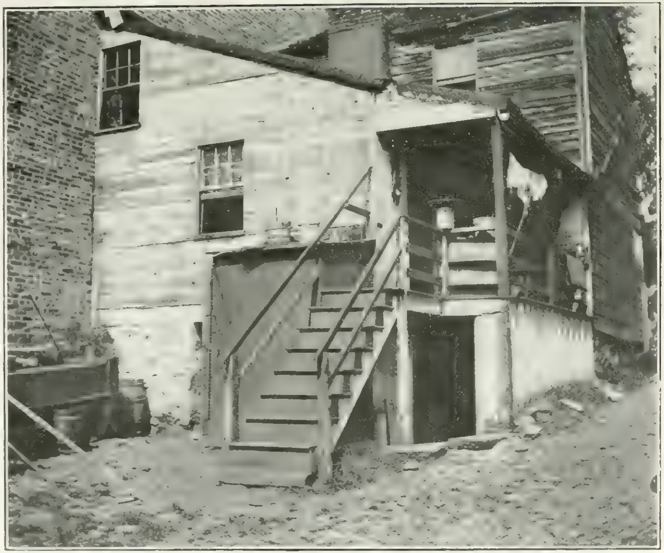
44. A DIRTY, UNTIDY MILK HOUSE.

45. A VERY NEAT, INEXPENSIVE, SMALL, BOTTLING ROOM.

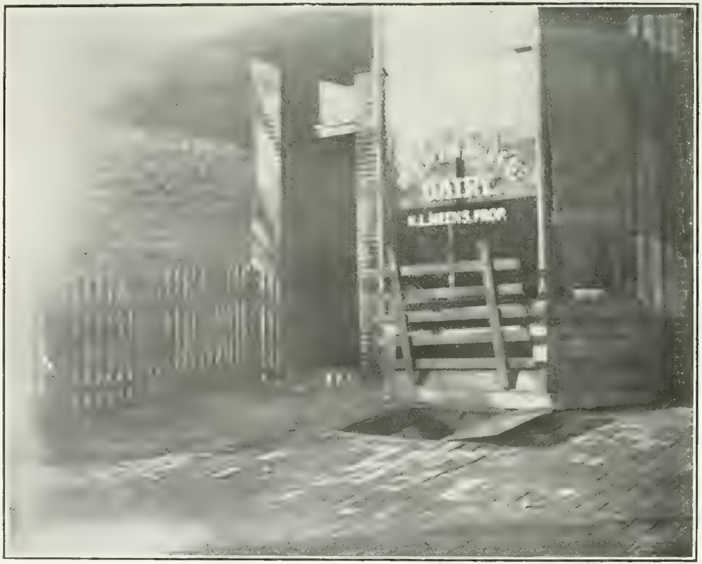


46. A MILK ROOM WITH ROUGH WALLS AND POORLY LOCATED TANK.

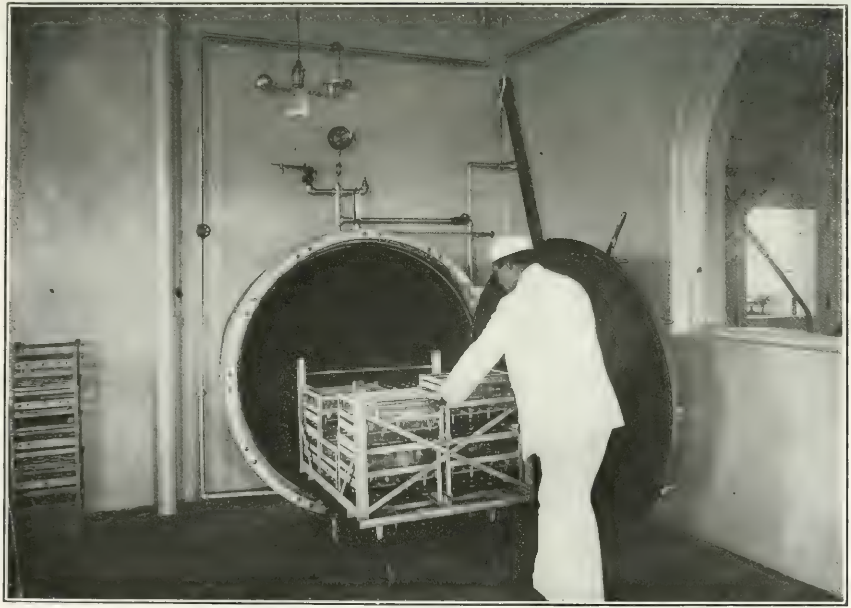
47. CHILDREN INTRUSTED WITH THE IMPORTANT WORK OF WASHING MILK BOTTLES.



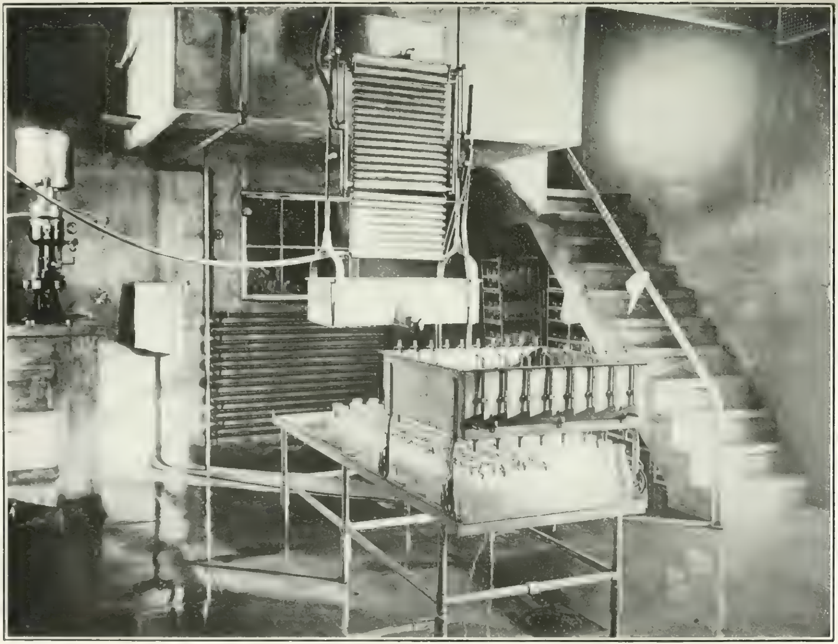
18. ENTRANCE TO DAIRY IS DOOR UNDER PIAZZA, BASEMENT OF RESIDENCE.



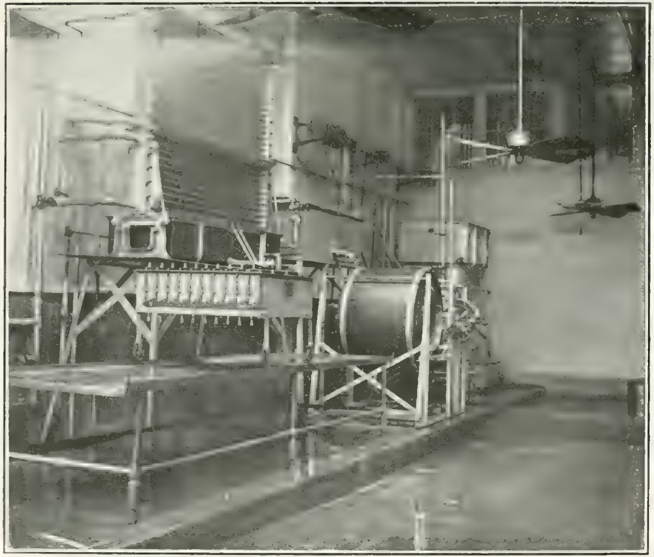
49. DAIRY ROOM IN CELLAR, UNDER STAIRS. NO LIGHT, NO VENTILATION.



50. A STERILIZATION OVEN USED IN BEST BOTTLING PLANTS.



51. BOTTLING ROOM IN A HIGH-CLASS CITY DAIRY.



52. A MODERN HIGH-CLASS PASTEURIZING PLANT.

An average of 13 experiments at Storrs station showed the following results:

Bacteria in milk from unwiped udders per c. c	7,058
Bacteria in milk from wiped udders per c. c	716
Decrease due to wiping	6,342

Fig. 30 is shown in contrast to Fig. 29 in order to illustrate a simple and inexpensive method of cleaning cows preparatory to milking and to show more strongly the desirability of such work. It is not probable that cows handled as those shown in Fig. 30 would ever get into the condition of the one shown in Fig. 29, but the proprietor of this clean dairy considered it necessary to wash and wipe the udders and flanks before every milking, in this manner. He is taking no risks in lives of infants who may be fed on milk from this dairy.

Figs. 35 and 36 show a clean exterior and interior. In such a place the problem of producing clean milk is much simplified, because the surroundings are in clean, sanitary condition. The filthiness shown in Fig. 29 could not exist.

The milker may not be cleanly in person or dress; he may have that most filthy of habits, milking with wet hands. The hands are usually wet by milking a few streams over them and kept wet by repeating the operation from time to time. The filth on the udder will ooze out under and through the fingers and drip into the pail. No illustration could be obtained showing this condition but it is frequently met with in inspection work.

Milkers too often wear the clothing that has done duty for every other work about the farm. Such clothing may contain dirt from the hog pen, the chicken coop, the horse barn, or the swill barrel, and is entirely unfit to be worn during milking. A clean white milking suit has a twofold effect. It will not of itself contaminate the milk, and if the milker is required to keep such a suit clean, he must of necessity keep everything with which he comes in contact clean. Compare the appearance of the milker shown in Fig. 37 with that in Fig. 29. A milker can not sit down to a dirty cow and keep himself or the milk clean.

The difference in results between different milkers working under the same conditions is strikingly illustrated by Stocking. The average of 19 tests in which 2 milkers who had had no training in dairy sanitation and 1 milker a graduate of the Connecticut Agricultural College showed 17,105 bacteria per cubic centimeter for the untrained men and 2,155 for the trained man. The only difference between the men was the knowledge of what constituted contamination gained by the college graduate, who was a student of bacteriology.

This example well illustrates the difficulty encountered in securing clean milk by means of police regulations only. Education must go first, and the police authority used only in those cases where the dairyman persists in wilfully violating his own knowledge in preparing his milk for market.

Any superficial compliance with police regulations becomes a farce unless the dairyman understands the principle back of such regulations. As an example:

Fig. 38 gives a graphic illustration of blind compliance with such regulations. A city ordinance required that there must be so many feet of glass in every stable. The barn in the photograph was one of the usual type of barn found in Pennsylvania and Maryland. The dairyman put in the required amount of glass behind the shutters! This was an extreme case, but the example shows the probable outcome of enforcing regulations without giving instruction as to their purpose. Fig. 39 illustrates the same thing. The dairyman was required to provide a milk house. Having no knowledge of the purpose of such a house he followed the idea that appealed most to him—that of convenience. This led to the ridiculous situation of his using a section of the hog house to keep his milk in. The skim and surplus milk was thus easily disposed of, but think of the condition of the milk that was sent to market after having been kept for some hours in such a place.

But little improvement will come through regulations requiring clean cows, clean milkers, and clean methods of milking and handling the milk unless the dairyman understands the object of such regulations and the effect they will have on his work. The officers in charge of inspection must be teachers first and policemen only when they find that the dairyman will not live up to the instructions given him and his knowledge of what is right.

MILK UTENSILS.

The milk pail should be made so as to reduce to a minimum the amount of dirt and hair that can get into it during the operation of milking. Fig. 40 shows various types of pails. The wide top is in most common use and is most objectionable. The narrow top in some form or other will undoubtedly in time replace the wide top. Pails and all other vessels designed to hold milk should be seamless, if possible, and where seams must occur they should be flushed full and smooth with solder. There should be no place either inside or out that can not be reached with the brush in washing. Heavily tinned utensils are recognized as the best for milk purposes. Wood, galvanized iron, or any material that is rough or porous is unfit for milk vessels.

CLEANING MILK UTENSILS.

No part of the dairy work is more important than the cleaning of the milk utensils, or is so often neglected. It can not be too strongly emphasized that dairy utensils must, after the milk is washed from the surface with warm water, be scalded with boiling water or steam. Nothing short of this will insure clean milk.

MILK HOUSES.

Milk must be removed at once from the barn to a clean place for cooling. The milk house must be provided with ample supply of hot and cold water, the necessary cooler, and other apparatus and supplies for handling milk. The surroundings of the milk house should be neat and clean and the air at all times free from objectionable odors. The following illustrations show good and bad conditions as found in inspection work. Figs. 41 and 42 are the exterior and interior views of a cheap but good milk house where milk is sold from the farm in bulk. Cement finish on the interior would be better than the wood, but the success of this place was due to the scrupulous cleanliness observed, and under these conditions the wood was unobjectionable. Figs. 43 and 44 are two very bad conditions. Fig. 43 shows the turkeys roosting in and around the milk house and on the milk utensils. The building is so open that no protection is afforded from dirt and intruders of all kinds. Fig. 44 shows very untidy surroundings. The barrels of trash and old wheelbarrows clutter up the yard and make it impossible to keep the premises clean. The door is off its hinges and altogether the place is unfit for the handling of milk. Fig. 45 shows the interior of a small bottling plant. Note the cleanliness of the attendant and the place in general. There is no expensive machinery, but the milk sold from this place is pure. Fig. 46 is the interior of a farm dairy room where milk is sold at wholesale. The room has an untidy appearance. The tank is located so that it will collect all the dirt from the floor. The position of the cans makes it more than probable that dirt will blow or be swept into them from the floor. The ceiling and walls can not be kept clean, there being too many places to catch dust and cobwebs. It will be noticed that the covers of the cans are not on tight. This is a practice quite common and is due to an idea that there must be some way for the bad odors to escape. If there are bad odors that should escape it is evidence that the milk is not clean. Clean milk needs no other aeration than that given it during the process of cooling.

CARING FOR THE MILK.

The bacterial content of the milk at any time depends upon the age of the milk, the initial number of bacteria introduced through process of milking and handling, and the temperature at which the milk has been kept. Consequently clean milk, quick cooling, and short time between milking and consumption are very important factors in securing pure milk.

A careful survey of the milk supply of a number of cities indicates that not enough attention is paid to these factors either by producers or distributors or by the inspection authorities. Milk should be cooled immediately and kept cool until it is consumed. From the farm to the consumer often several agencies are employed. Hauling to the depot, holding at shipping point, transportation on the cars, and the handling in the city milk plants are each steps in the process of supplying a city that need intelligent and conscientious care.

THE CITY DISTRIBUTING PLANT.

All that has been said about cleanliness, surroundings, and care in handling milk on the farm applies to the city plant where milk is received and distributed to the consumer. So far as insanitary surroundings are concerned it is usually the smaller dealer who is the greatest offender. He usually lacks facilities for scalding or sterilizing bottles and utensils. Fig. 47 shows a condition that is in many places too common. Bottles are washed in lukewarm water and no provision of any kind is made for sterilizing them. Children are intrusted with the work. Fig. 48 shows a condition somewhat similar, but the milk room is in the basement of a house in which the family lives. Family affairs and the handling of the milk are brought into too close proximity. The basement is dark and illy ventilated.

Fig. 49 is much worse, the trap door in the sidewalk is the only means of entrance for attendants, light, and ventilation. A leaky sewer pipe runs across the ceiling of the cellar. To bottle and sell milk from such a place should be a criminal offense.

Figs. 50 and 51 are excellent views of the sterilizing room and the bottling room of a high-class city dairy, and show a marked contrast to the preceding illustrations.

Fig. 52 shows the interior of a modern pasteurizing and bottling plant. It is ideal in every way for such work.

It may be said that to require conditions like those shown in Figs. 50, 51, and 52 would put all small dealers out of business. This is not necessarily true. There is no reason why a small dealer can not be as clean and careful in his work as a large one. Anyone handling milk in small quantities as shown in Figs. 47, 48, and 49 could maintain a place like that shown in Fig. 45, which is on a dairy farm. If they could not the health of the public demands that they quit the business.

THE SCORE-CARD SYSTEM OF INSPECTING DAIRIES.

For the past three years the Bureau of Animal Industry through the Dairy Division has been making a thorough investigation of the milk supply of a large number of cities of the country with a view of establishing a system of inspection and reporting on dairies that would be complete, comprehensive, and meet the needs of the public in improvement of the milk supply. Doctor Woodward, Health Officer of the District of Columbia, was the first to introduce a score-card system of reporting on dairies. A little later Prof. R. A. Pearson, of Cornell University, introduced a card for the same purpose. These cards had many good features, and if they had been generally adopted would have done much to improve the dairy conditions of the country.

The Department of Agriculture took up the work with the hope of extending the use of the score card and more thorough inspection thereby. After three years' work, scoring several thousand dairies in all parts of the country, a score card has been adopted which has been introduced in a more or less modified form, and is in use in about 60 of the larger cities of the country and in many smaller ones. The following are the forms of the present cards for farm dairies and city milk plants:

[United States Department of Agriculture, Bureau of Animal Industry, Dairy Division.]

Sanitary inspection of dairies.

[Adopted by the Official Dairy Instructors' Association.]

Owner or lessee of farm..

P. O. address........................ State........................

Total number of cows........................ Number milking........................

Gallons of milk produced daily..

Product is retailed by producer in..

Sold at wholesale to..

For milk supply of..

Permit No................ Date of inspection.., 19

Remarks..

....

....

(*Signed*)..

Inspector.

Detailed score.

Equipment.	Score.		Methods.	Score.	
	Perfect.	Allowed.		Perfect.	Allowed.
COWS.			COWS.		
Health...........	6	....	Cleanliness of cows.........	8	....
Apparently in good health.................. 1			STABLES.		
If tested with tuberculin once a year and no tuberculosis is found, or if tested once in six months and all reacting animals removed ... 5			Cleanliness of stables........	6	....
(If tested only once a year and reacting animals found and removed, 2.)			Floor..................... 2		
Comfort ..	2	..	Walls..................... 1		
Bedding 1			Ceiling and ledges....... 1		
Temperature of stable... 1			Mangers and partitions.. 1		
Food (clean and wholesome)..	2	....	Windows................ 1		
Water..	2	....	Stable air at milking time....	6	....
Clean and fresh 1			Barnyard clean and well drained...	2	....
Convenient and abundant.................... 1			Removal of manure daily to field or proper pit... ..	2	....
			(To 50 feet from stable, 1.)		
			MILK ROOM.		
			Cleanliness of milk room......	3	....

Detailed score—Continued.

Equipment.	Score. Perfect.	Score. Allowed.
STABLES.		
Location of stable	2	
Well drained........... 1		
Free from contaminating surroundings.......... 1		
Construction of stable	4	
Tight, sound floor and proper gutter.......... 2		
Smooth, tight walls and ceiling.......... 1		
Proper stall, tie, and manger.......... 1		
Light: Four sq. ft. of glass per cow	4	
(Three sq. ft., 3; 2 sq. ft., 2; 1 sq. ft., 1. Deduct for uneven distribution.)		
Ventilation: Automatic system	3	
(Adjustable windows, 1.)		
Cubic feet of space for cow: 500 to 1,000 feet	3	
(Less than 500 feet, 2; less than 400 feet, 1; less than 300 feet, 0; over 1,000 feet, 0.)		
UTENSILS.		
Construction and condition of utensils	1	
Water for cleaning	1	
(Clean, convenient, and abundant.)		
Small-top milking pail	3	
Facilities for hot water or steam	1	
(Should be in milk house, not in kitchen.)		
Milk cooler	1	
Clean milking suits	1	
MILK ROOM.		
Location of milk room	2	
Free from contaminating surroundings.......... 1		
Convenient.......... 1		
Construction of milk room	2	
Floor, walls, and ceiling . 1		
Light, ventilation, screens.... 1		
Total	40	

Methods.	Score. Perfect.	Score. Allowed.
UTENSILS AND MILKING.		
Care and cleanliness of utensils	8	
Thoroughly washed and sterilized in live steam for 30 minutes.......... 5		
(Thoroughly washed and placed over steam jet, 4; thoroughly washed and scalded with boiling water, 3; thoroughly washed, not scalded, 2.)		
Inverted in pure air..... 3		
Cleanliness of milking	9	
Clean, dry hands..... 3		
Udders washed and dried 6		
(Udders cleaned with moist cloth, 4; cleaned with dry cloth at least 15 minutes before milking, 1.)		
HANDLING THE MILK.		
Cleanliness of attendants	1	
Milk removed immediately from stable	2	
Prompt cooling (cooled immediately after milking each cow)	2	
Efficient cooling; below 50° F.	5	
(51° to 55°, 4; 56° to 60°, 2.)		
Storage; below 50° F.	3	
(51° to 55°, 2; 56° to 60°, 1.)		
Transportation; iced in summer	3	
(For jacket or wet blanket allow 2; dry blanket or covered wagon, 1.)		
Total	60	

Score for equipment...... + score for methods...... = ..., final score.

Note 1.—If any filthy condition is found, particularly dirty utensils, the total score shall be limited to 49.

Note 2.—If the water is exposed to dangerous contamination or there is evidence of the presence of a dangerous disease in animals or attendants, the score shall be 0.

[United States Department of Agriculture, Bureau of Animal Industry, Dairy Division.]

Sanitary inspection of city milk plants.

Owner or manager.......... Trade name..........

City.......... Street and No.......... State..........

Number of wagons.......... Gallons sold daily {Milk.......... Cream..........

Permit or license No.......... Date of inspection.........., 190

	Score.		Remarks.
	Perfect.	Allowed.	
Milk room.			
Location	10		
Construction			
Floor (3)			
Walls and ceiling (3)	10		
Drainage (4)			
Cleanliness	15		
Light and ventilation	10		
Equipment –			
Arrangement (3)			
Construction			
Sanitary (2)	15		
Durability (2)			
Condition (3)			
Cleanliness (5)			
Milk.			
Handling (12)	20		
Storage (8)			
Sales room.			
Location (2)			
Construction (2)	10		
Equipment (2)			
Cleanliness (4)			
Wagons.			
General appearance (2)			
Protection of product (3)	10		
Cleanliness (5)			
Total	100		

Sanitary conditions are—Excellent.......... Good............ Fair.......... Poor..............

Suggestions by inspector..

Signed..

Inspector.

Sanitary inspection of city milk plants (reverse side).

DIRECTIONS FOR SCORING.

MILK ROOM.

Location.—If not connected by door with any other building, and surroundings are good, 10; when connected with other rooms, such as kitchens, stables, etc., make deductions according to conditions.

Construction. If good cement floor, and tight, smooth walls and ceiling, and good drainage, allow 10, deduct for cracked or decayed floors, imperfect wall and ceiling, etc.

Cleanliness. If perfectly clean throughout, allow 15; deduct for bad odors, unclean floor and walls, cobwebs, unnecessary articles stored in room, etc.

Light and ventilation. If window space is equivalent to 15% or more of the floor space, allow 5; deduc 1 point for every 3% less than the above amount.

Equipment.

Arrangement.—Allow 3 points for good arrangement; if some of the equipment is out of doors or so placed that it can not be readily cleaned, make deductions according to circumstances.

Condition.—If in good repair, allow 4 points; make deductions for rusty, worn-out, or damaged apparatus.

Construction.

Sanitary: If seams are smooth, and all parts can be readily cleaned, allow 2. Deduct for poor construction, from sanitary standpoint.

Durability: If made strong and of good material, allow 2. Deduct for light construction and poor material.

Cleanliness.—If perfectly clean, allow 8 points; make deductions according to amount of apparatus improperly cleaned.

MILK.

Handling.—If milk is promptly cooled to 50° F. or lower, allow 12 points; or if pasteurized at a temperature of 149° F. or above and promptly cooled to 50° or lower, allow 12 points. Deduct 1 point for every 2° above 50°. If milk is pasteurized imperfectly, deduct 6 points. If milk is improperly bottled or otherwise poorly handled, make deductions accordingly.

Storage.—If stored at a temperature of 45° F. or below, allow 8 points. Deduct 1 point for every 2° above 45°.

SALES ROOM.

Location.—If exterior surroundings are good and building is not connected with any other under undesirable conditions, allow 2; for fair conditions, allow 1; poor conditions, 0.

Construction.—If constructed of material that can be kept clean and sanitary, allow 2; for fair construction, allow 1; poor construction, 0.

Equipment.—If well equipped with everything necessary for the trade, allow 2; fair equipment, 1; poor equipment, 0.

Cleanliness.—If perfectly clean, allow 4 points; if conditions are good, 2; fair, 1; poor, 0.

WAGONS.

General appearance.—If painted and in good repair, allow 2 points; for fair condition, 1; poor, 0.

Protection of product.—If product is iced, allow 3 points; well protected but not iced, 1; no protection, 0.

Cleanliness.—If perfectly clean, allow 5; good, 3; fair, 2; poor, 0.

The use of these cards enables a more perfect study of conditions in any city. The results so reported are comparable and can be analyzed with greater ease and accuracy.

The application of the system to the District of Columbia and vicinity shows the following conditions:

Eight hundred and eighty-six dairies were given complete scores, of which 526 were in Maryland and 294 in Virginia and 66 in the District.

The average scores are as follows:

	Per cent.
District of Columbia	53.44
Virginia	51.71
Maryland	40.42
Average of all	45.03

The greater bulk of milk comes from four counties in which the average score, respectively, is as follows:

	Per cent.
Loudoun County, Va	55.00
Fairfax County, Va	53.25
Montgomery County, Md	42.77
Frederick County, Md	38.47

The following table shows the number and per cent of the whole umber in each of the three sections, District of Columbia, Maryland, nd Virginia—scoring between 0–10, 10 and 20, 20 and 30, etc.:

Scoring from	Virginia.		Maryland.		District of Columbia.	
	Number.	Per cent of all.	Number.	Per cent of all.	Number.	Per cent of all.
0 to 10						
0 to 20			4	0.76		
20 to 30	7	2.18	76	14.44	2	3.02
30 to 40	44	14.96	173	32.85	7	10.60
40 to 50	83	28.23	164	31.18	13	19.69
50 to 60	89	30.20	74	14.01	21	31.81
60 to 70	53	18.02	22	4.18	13	19.69
70 to 80	14	4.42	10	1.90	9	13.63
80 to 90	3	1.02	3	.55	1	1.51
90 to 100	1	.34				

The inspection of the city milk plants showed a better condition of affairs in some particulars than the inspection of the farms.

Seventy-four establishments showed an average score of 72.58 per cent; 4 per cent scored 90 or above; 16 per cent scored in the eighties; 49 per cent scored in the seventies; 25 per cent scored in the sixties; 6 per cent scored in the fifties.

The average rating of the dairies and city milk plants in the vicinity of Washington does not differ materially from that in other parts of the country. The cities that have adopted the score-card system and are regularly following it up are reporting dairy conditions constantly improving. Dealers take advantage of the situation and bid up on the high-scoring dairies and thus encourage better work on the part of the farmer. The dairymen want a high score and by studying the points in the score card are able to improve their conditions. On subsequent inspection they get a better score in almost every instance.

A number of cities publish from time to time in the local papers or through boards of health circulars the complete results of the score. This publicity has a very stimulating effect in inducing the dairymen and milk dealers to improve their conditions.

Perhaps not the least valuable part of the score-card system is the demand it makes for better and more competent inspectors. It has usually resulted in the dissatisfaction of inspectors with their previous work. The study of the premises in detail with the view of fixing the exact value of conditions requires better and more conscientious work. Wherever the system has failed, and there have been but one or two such, it has failed because the inspector did not relish the comparison of the results with his previous work.

The photographs shown in this article were all taken in the course of inspection work. Many of them were taken in the vicinity of Washington, but duplicates of these places may be found in all parts of the country. They show that more rigid inspection is absolutely necessary, that competent inspectors must be employed to instruct, and where instruction is not sufficient, to demand a better state of affairs. Laws and ordinances must be strengthened and the public educated to demand clean milk from clean dairies.

One important item that must be borne in mind is the fact that to change these conditions must create some expense on the producer and the consumer must expect to foot the bills.

A prominent veterinarian in Kansas City, Mo., recently said in connection with the milk supply of that city, that the prevailing price of milk was based on a system which required only that the solid and coarsest dirt be strained out. If the consumer wanted milk that had been kept free from such contamination he would have to pay for the added cost of production.

In order to carry into effect such a system of inspection it is recommended that there be an inspector for approximately every 100 dairy farms. These inspectors should be skilled in all questions pertaining to the production and distribution of milk. Five of every ten inspectors so employed should be skilled veterinarians and the other five should have had training in a good dairy school or have had experience which would be the equivalent of such training.

Inspectors should devote their entire time to the work of inspection and should not be allowed to do outside work that in any way relates to the business of inspection. There should be a chief inspector whose duty it is to supervise all work of inspection; he should be responsible to the health officer. The health officer, or board of health, should have full power to make rules and regulations and enforce the same so as to safeguard the health of the community from a contaminated milk supply either through carelessness, ignorance, or malicious intent.

The following suggestions might well be distributed by the health officer and the requirement be made that they be posted in a conspicuous place in every barn, dairy house, and city milk plant:

TWENTY-ONE SUGGESTIONS.

THE COWS.

1. Have the herd examined frequently by a skilled veterinarian. Promptly remove any animals suspected of being in bad health. Never add an animal to the herd until certain it is free from disease, especially tuberculosis.

2. Never allow a cow to be excited by hard driving, abuse, loud talking, or unnecessary disturbances; do not unduly expose her to cold or storms.

3. Clean the entire body of the cow daily. Hair in the region of the udder should be kept short. Wipe the udder and surrounding parts with a clean, damp cloth before milking.

4. Do not allow any strong flavored feed, such as garlic, cabbage, or turnips, to be eaten except immediately after milking.

5. Salt should always be accessible.

6. Radical changes in feed should be made gradually.

7. Have fresh, pure water in abundance, easy of access, and not too cold.

THE STABLES.

8. Dairy cattle should be kept in a stable where no other animals are housed, preferably without cellar or storage loft. Stable should be light (4 square feet of glass per cow) and dry, with at least 500 cubic feet of air to each animal. It should have air inlets and outlets, so arranged as to give good ventilation without drafts of air on cows. The presence of flies may be reduced by darkening the stable and removing the manure as directed below.

9. The floor, walls, and ceilings of the stable should be tight, walls and ceilings being kept free of cobwebs and whitewashed twice a year. There should be as few dust-catching ledges and projections as possible.

10. Allow no musty or dirty litter or strong-smelling material in the stable. Store manure under cover at least 40 feet from the stable in a dark place. Use land plaster daily in gutter and on floor.

THE MILK HOUSE.

11. Cans should not remain in the stable while being filled. Remove the milk of each cow at once from the stable to a clean room; strain immediately through cotton flannel or absorbent cotton; cool to 50° F. as soon as strained, store at 50° F. or lower. All milk houses should be screened.

12. Milk utensils should be made of metal, with all joints smoothly soldered, or, when possible, should be made of stamped metal. Never allow utensils to become rusty or rough inside. Use milk utensils for nothing but handling, storing, or delivering milk.

13. To clean dairy utensils use pure water only. First rinse the utensils in warm water; then wash inside and out in hot water in which a cleansing material has been dissolved; rinse again; sterilize with boiling water or steam; then keep inverted in pure air that may have ready access, and sun if possible, until ready for use.

MILKING AND HANDLING MILK.

14. The milker should wash his hands immediately before milking and should milk with dry hands. He should wear a clean outer garment, which should be kept in a clean place when not in use. Tobacco should not be used while milking.

15. In milking be quiet, quick, clean, and thorough. Commence milking at the same hour every morning and evening and milk the cows in the same order.

16. If any part of the milk is bloody, stringy, or unnatural in appearance, or if by accident dirt gets into the milk pail, the whole mess should be rejected.

17. Weigh and record the milk given by each cow.

18. Never mix warm milk with that which has been cooled, and do not allow milk to freeze.

19. Feed no dry, dusty feed just previous to milking.

20. Persons suffering from any disease, or who have been exposed to a contagious disease, must remain away from the cows and the milk.

21. It is needless to say that the shorter the time between the production of milk and its delivery, and between delivery and use, the better will be the quality of the milk.

17. SANITARY WATER SUPPLIES FOR DAIRY FARMS.

SANITARY WATER SUPPLIES FOR DAIRY FARMS.

By B. Meade Bolton, M. D.,

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The importance of sanitary water supplies for farms is not confined to the farmer himself and his immediate family, but it extends to the whole public. All products of the farm with which water comes in contact may be rendered dangerous to health by polluted water. All fruits and vegetables and receptacles used for collecting and shipping food and the hands of those who collect and pack farm produce may be infected by washing in impure water. The infection in this case arises from the bacteria contained in the water, and after the bacteria are once conveyed to the food they increase very rapidly under conditions favorable to them. Many kinds of food furnish a favorable soil for the growth of bacteria, and this is particularly the case with milk. Milk constitutes in fact one of the very best media for the development of many kinds of bacteria. Even with great care and cleanliness in milking, the bacteria which get into the milk quickly multiply to many thousands, unless the milk is kept cold. Bacterial contamination of milk arises not only from dust, hair, and filth at milking, but also comes from polluted water used by milkers for washing the hands, the udders, the milk pails and cans. The drops remaining in the can after rinsing with impure water are sufficient to contaminate all the milk put into the can, and the bacteria introduced into the milk in this way multiply rapidly, unless the milk is kept very cold. The danger from polluted milk is not only that there may be microbes present which may cause special diseases, such as typhoid fever and scarlet fever, but also that many bacteria cause changes in the milk which make it injurious to health, particularly injurious to children. In this case the bacteria themselves may be of such a kind as not to produce disease if taken into the stomach alone, but they may nevertheless change the milk so as to make it to all intents a poison.

The same thing is true more or less with all food, particularly with food which is eaten raw, but it is especially the case with milk.

It may not be out of place to correct an erroneous idea which seems quite prevalent in regard to milk contamination through polluted water. The belief among farmers appears to be quite widespread that milk may become contaminated by the impure water drunk by the cows, the conception being that in some way bacteria pass from the stomach of the cow through the udder into the milk.

There seems no good reason for believing that this takes place. It is true that milk becomes tainted by garlic and weeds which the cow eats, but this is a very different matter from the passage of bacteria from the cow's stomach into the milk. The danger from bacteria in milk arises mainly if not wholly from the use of unclean vessels, and from slovenly methods of handling the milk in the case of healthy cows. It is true that if the cow is diseased, particularly if tuberculosis exists or disease of the udder, bacteria may get into the milk from the cow.

While it is true as just stated that bacteria from foul water does not pass directly from the stomach of the cow into the milk, it is nevertheless important for the health of the cow to have an abundance of pure water to drink.

REQUIREMENTS OF A SANITARY WATER SUPPLY.

The three factors necessary for a sanitary water supply are purity, abundance, and convenience. The most important of these factors, and that which has received most consideration as a rule is purity. It is the most obvious of the three in importance, since people naturally prefer clean, pure water, and they are generally educated to the dangers arising from polluted water as a possible source of infection. Hygienic examinations of water supplies often begin and end with a determination of bacteriological or chemical contamination to the neglect of the questions of proper location, abundance, and convenience. Now, while it is quite proper that the purity of the water should receive the first consideration, the other two factors can not be safely ignored. The water may be pure and sufficient for drinking purposes, and yet not in sufficient amount for cleanliness. For sanitary purposes it is essential that the water should be in such quantity that there is no need for stinting in any direction. It is essential to have abundance for personal cleanliness, for the laundry, for washing the utensils of the kitchen or dairy, and for the premises generally. It should be abundant at all seasons. The importance of the unrestricted use of water is so great that some hygienists for this reason condemn the use of water meters in private houses in cities with a central water supply, because many people are apt to stint themselves if the water is paid for by the amount used.

Convenience is probably the least important factor, but it is nevertheless essential for a sanitary water supply. It seems from the result of the inspection of about 300 farms around Washington that his requirement is more neglected than the matter of pollution or of abundance. Most farmers take pride in what they regard as the purity and abundance of their water supply. Each one in the neighborhood will frequently boast of his spring or well in these respects, but many of them will year after year haul the water up in a bucket out of an open well or pump the water by hand into a pail or bring it by hand up hill from the spring. Where such exertion as this is necessary in good and bad weather alike persons will resort to economy in the use of water for cleaning purposes at least.

No one rule for preserving health is more important than cleanliness, the frequent bath, clean underwear, cleanliness of vessels used for food, particularly cleanliness of receptacles for milk, cleanliness of dwelling and stable. Nothing is so conducive to cleanliness as an abundant and convenient supply of clean water and anything which facilitates the unrestricted use of pure water is in itself a hygienic measure.

SOURCES OF WATER SUPPLY.

Of the water which falls to the earth as rain, hail, or snow, a large part is evaporated and taken back up into the air. Of the rest a part runs off to feed the brooks and rivers, and a part sinks into the soil. It is mainly the portion which sinks into the ground which is of interest as a source of domestic water supply for the farm, for it feeds the springs and wells. It percolates through the soil until it reaches the so-called "water table" which is a more or less porous layer of gravel or sand lying upon an impervious stratum of clay or rock (see fig. 53).

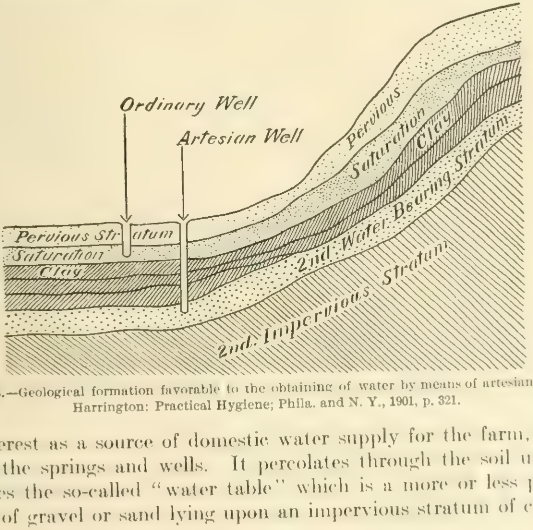


FIG. 53.—Geological formation favorable to the obtaining of water by means of artesian wells. Harrington: Practical Hygiene; Phila. and N. Y., 1901, p. 321.

The water table follows the dip of the rock or clay layer and is consequently to be found at various depths or it may crop out on the surface. Where it crops up it forms a spring where it is tapped by a shaft it furnishes the water for a well. If this water table lies between two impervious layers, and if the point at which the well is sunk is at a lower level than any part of the water table, the water flows out and constitutes an artesian well. In order that the water may come out at the top of the well it is of course essential that the water table should lie between two impervious layers. The upper of the two may, however, be at the surface of the ground. A sub-artesian well is one in which the water comes up near the top of the shaft.

SOURCES OF POLLUTION.

Water takes up some part of everything with which it comes in contact. Some things, like common salt and potash, as everyone knows, are readily dissolved in water, while many other substances are dissolved in very small traces; but the solvent action of water even on the hardest stone may be noticed in time. Not only solid substances, but gases and liquids, as well as living micro-organisms, microscopic plants and animals, and minute particles of dust are all taken up by water. From this fact it is evident that everything with which it comes in contact from the time it leaves the ocean as clouds to the time it returns to the ocean as rivers is taken up by the water to a greater or less extent. In other words everything which is found either dissolved or floating in water except such substances as are introduced directly, either intentionally or by accident, is derived from the air or soil through which the water passes on its way from the ocean and back again. On its way through the air the water takes up various gases—oxygen, nitrogen, carbon dioxide, ammonia, and other substances of this nature as well as fine dust particles and bacteria. On its passage through the soil it dissolves various minerals from the rocks, such as lime and magnesia, and if the soil is polluted in any way, it takes up whatever it can dissolve of the pollution. In the upper layers of the soil the water also comes in contact with bacteria which cause its contamination.

Many of the substances taken up by the water are harmless or even beneficial, others are undesirable, while others again may be harmful in themselves or indicative of former pollution. The nitrogen and ammonia from the air are probably without significance from a sanitary point of view, though these may be of some value in a different direction, as a source of food for growing plants. The oxygen and carbon dioxide serve a useful purpose in giving life and sparkle to the water and in this way impart an agreeable taste. The bacteria which the water takes from the air are probably seldom of any significance, though it is true that occasionally bacteria of certain diseases have been found floating in the air and these may be taken up by the water; still this is probably not frequent in rural districts at least.

The mineral matter taken up from the soil, particularly the salts of lime and magnesia, make the water "hard," and although this does not affect the health of those who take the water, unless the minerals be present in large amounts, it makes the water less suitable for purposes of cleanliness. The presence of sewage is of course an indication at least that the water may be injurious to health; for as everyone knows outbreaks of typhoid fever and of disorders of the bowels have been frequently traced to water that was polluted with sewage.

The substances other than the bacteria taken up by the water are not themselves injurious, but they may be more or less significant of pollution. Since the disease-producing property of polluted water is due to the bacteria which it contains, it would be obviously of the very greatest advantage to be able in any given case to detect the presence of the pathogenic organism concerned. It goes without saying that it would be very desirable to be able to state in a given case that the germ of typhoid fever or of scarlet fever or of dysentery were or were not present in the water. Unfortunately our knowledge has not yet been advanced to the point at which this is possible. There are many instances on record where epidemics of typhoid fever have been traced to certain water supplies, and yet the most painstaking search has failed to show the presence of the bacterium which is generally regarded as the cause of typhoid fever. In fact the cases are comparatively rare in which this organism has been claimed to be detected in water which seemed beyond doubt to be the cause of typhoid fever. The most that can be accomplished by chemical analysis and bacteriological examination of water at present is to show with more or less certainty the presence of pollution with sewage. It is not possible to state positively as a result of these tests that any given specimen of water, even though it contain sewage, will produce typhoid fever or other gastro-intestinal disorders when taken into the stomach. But it is quite possible by these means to show that water is more or less polluted and consequently a menace to health. This is after all probably as useful from a practical sanitary point of view as the ability to detect the specific organism of disease would be. If by any means it can be shown that a water supply is polluted or even suspicious this is reason sufficient for taking steps to prevent the continuance of the pollution or failing in this to condemn and close the source.

It is rarely possible to form a just opinion simply by the examination of samples of water sent from a distance, for there are other factors to be taken into consideration. The decision in regard to the potability of water in any given case is a matter often requiring very careful consideration of the results of analysis, along with a sanitary survey and a knowledge of the normal standards for pure water in the section from which the sample is obtained. In forming a judgment the inspection of the premises and the normal standard of other waters in the same region coming from demonstrably unpolluted sources must be taken into account. Injustice and hardship have unquestionably resulted from the condemnation of water supplies on chemical and bacteriological examinations alone, without regard to local conditions. The standards of purity should be established for each neighborhood for itself by the analysis of samples from several supplies of unpolluted water in the locality. It by no means follows that what has been found as a standard for Massachusetts or Michigan can be used as a standard for Maryland and Virginia, nor that the standard for even one section in Virginia or Maryland can be used as a standard for another part of the same States. In fact, of some 150 water supplies on farms in Virginia recently examined, it was very apparent that certain substances regarded as indications of pollution were present in unquestionably uncontaminated waters in some localities in greatly larger amounts than pure water in other localities in the same section. It does not seem justifiable in the light of the data obtained in the examination of these supplies to take the standards laid down in the books as a mechanical and inelastic measure of the purity of these waters. In the statements just made the chemical examination of water was specially had in mind, but the statements apply equally well to bacteriological examination, particularly to bacteriological examination made of water shipped from a distance where judgment is pronounced without reference to local conditions or other modifying circumstances.

As in the case of the chemical examination, the bacteriological examination as a rule merely indicates the probabilities in regard to pollution; it does not give in most cases at least positive information as to the presence or absence of organisms which would cause this, that, or the other disease. It is, moreover, a matter of experience that the results of a bacteriological examination made at one time may differ very decidedly from that made at another time of the same water under apparently the same conditions. In regard to the detection of *Bacillus coli communis*, which is at present regarded by many as a more or less trustworthy indication of contamination, recent examination of the water supplies in Virginia has shown that this organism was present at one time and not present at another in the same water supply.

The significance of the colon bacillus in the dairy water supplies will be discussed in a different paper. It is merely referred to here or the sake of illustrating the variations which may occur in the esults of a bacteriological examination of water.

What has just been said in regard to the chemical and bacteriological examination of water does not apply of course to the applicaion of chemical and bacteriological examinations in general. On he contrary, it is possible to detect specific causes of disease by these means in many cases. One need only recall the value of the hemical and bacteriological examinations of urine in diseases of the kidney and bladder, and of the value of bacteriological examinations in cases of suspected tuberculosis and diphtheria. But on the other hand while such examinations of water supplies may be of very great value, they give us only the probabilities in the case, and these probabilities are open to some difference of opinion as to their weight. The more experience the observer has had the less inclined he is to make arbitrary standards and the more capable is he of forming a correct judgment in his interpretation of the results of his examination. In the examples given above of the detection of kidney disease and of the specific bacteria in diphtheria and tuberculosis, competent observers will all readily agree in the interpretation of results. In the matter of the probability of pollution of water there is more room for difference of opinion from results of analysis. The various statements made in text-books and in monographs on hygienic water analysis give ample evidence of the want of uniformity of opinion in this respect. It would seem essential in all cases to establish a standard of purity for the region of country from which any specimen of water under consideration comes.

There are competent observers with abundant experience who are inclined to question the value of chemical and bacteriological water analysis *in toto*, and in view of the arbitrary and mechanical manner in which the results of these analyses are sometimes interpreted this attitude is justified to some extent. It would seem, however, that after the establishment of normal standards for a given locality such analyses are useful if they are checked by intelligent consideration of all the conditions entering into the case but no hard and fast rules can be applied.

PURIFICATION OF WATER IN THE SOIL.

While water in its passage through the air and through the soil becomes contaminated with bacteria which may cause special disease or disturbance of digestion, it also undergoes on the other hand a process of purification, consisting in a filtration of the particles held in suspension. It has been found that at a comparatively short distance below the surface, 4 or 5 feet, there are frequently but few bacteria present in the ground, and the water which percolates through the soil, although it becomes contaminated in the upper layers, it is rid of bacteria on its passage farther downward. Deep ground water usually contains few bacteria, but it may become contaminated when it is tapped for a well. Of course if the layer of soil through which the water percolates on its way to the water table is saturated with filth some of the pollution may be carried down, particularly if the layer of soil is not thick.

PROTECTION FROM POLLUTION.

The water supplies of farms consist of wells, springs, and cisterns. A recent inspection of the water supplies of some 300 dairies in Maryland and Virginia showed that wells are used much oftener on these farms than either of the other two. The proportions are about 5 wells to 3 springs to 1 cistern.

Since, as has been stated, the sources of pollution are the entrance of sewage or other impurities through cracks and crevices or through a porous soil that has become saturated or finally by the intentional or accidental introduction of impurities, it is necessary to guard against each of these sources.

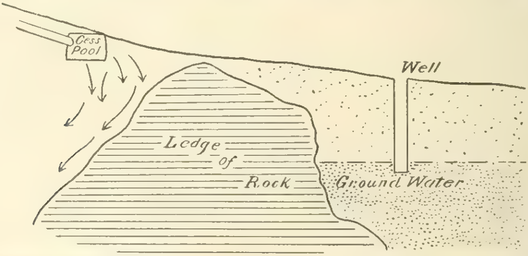


FIG. 54. Showing how a cesspool located on high ground may fail to pollute a well lower down. Harrington: Practical Hygiene; Phila. and N. Y., 1901, p. 324.

WELLS.

To guard against the pollution of wells the location is of importance. Where it is possible the ground should slope away naturally on all sides, and the pump should be on top of a mound which should be well sodded or cemented all around. Sources of domestic or of other pollution should be separated from the well by an impervious layer below ground to avoid the danger of pollution from seepage.

Figure 54 shows how a stratum of rock may protect a well from pollution, even where the drainage is toward the well.

The following figure shows how a cesspool may pollute a well, even though the cesspool is at a lower level than the top of the well:

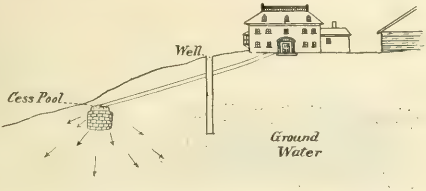


FIG. 55.—Showing pollution of a well by a cesspool situated on a lower level than the top of the well. Harrington: Practical Hygiene; Phila. and N. Y., 1901, p. 324.

The ground immediately around the well should be protected from stray animals by a fence or otherwise. The shaft of the well should be thoroughly tight and for this reason the use of terra cotta tiles or metal pipe, for the shaft is to be preferred to walling up with bricks and mortar. In any event the space immediately around the shaft proper should be puddled with clay or cement, or, as advised by Koch, have the upper part packed around with sand. The use of open wells or even the use of chain pumps is not to be recommended, since they are more or less liable to pollution from the introduction of impurities down the shaft. In all cases the well should be guarded by a tight coping and cover. A device sometimes resorted to, and which is an advantage, is to cover over the well with a tight cover and to place the pump to one side of the well shaft with an elbow connection.

A form of well, known variously as the tube, or driven, or Norton, or Abyssinian well, is good from a sanitary point of view. It consists merely of an iron pipe screwed together in sections driven down to the water-bearing layer. The lowest section of pipe is armed with a point and is perforated with a number of holes. In a well of this character there is no danger from seepage into the shaft and it is cheaply and quickly constructed. In case one such well fails to furnish sufficient water others can be driven alongside and all connected with one pump.

Every precaution should be taken to prevent the contents of the cesspool soaking into the soil, for even if the cesspool is at a distance from the well the ground between will eventually become saturated and fail to act as a filter. As stated above, the presence of an impervious stratum between the well and the cesspool is a good protection, but where such a condition does not exist the cesspool should be made water-tight. The crude methods of sewage disposal still quite commonly in vogue in the country lead to a continual menace of polluting the water supply.

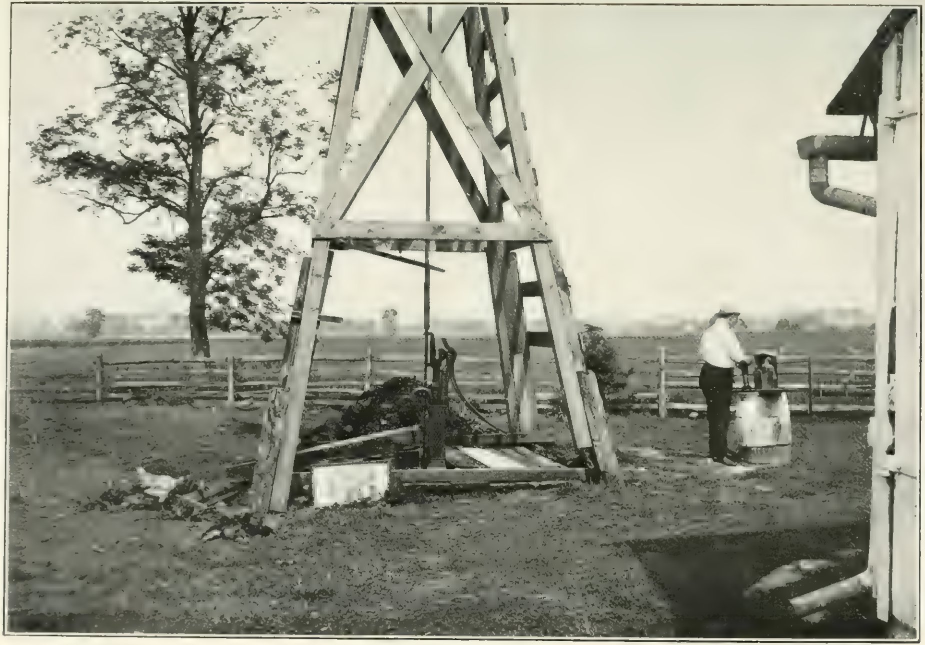
The photograph, No. 56, is of a well which is imperfectly guarded against pollution and with very slovenly surroundings. The situation of the well in this case is good. It lies at a considerably higher level than the barnyard which is below, and shown in the left-hand corner of the picture, and is separated from the well by a ledge of rock. The domestic sources of pollution lie to the right and are several hundred feet away. The building shown at the right-hand corner of the picture is a wood shed. The well is only about 7 feet deep, but it is bored into the solid rock and in spite of its want of depth, there would appear no good reason why it should not be made to fulfill the requirements of a sanitary supply, but when it was inspected it was found to have a loose coping and there was no provision against pollution due to stray animals.

Photograph No. 57 shows a well bored into solid rock, and although it is only 16 feet deep it would appear to be well protected from any source of contamination. Besides the protection afforded by the natural rock the curb and cover are tight, and moreover the cover is given a slant so as to shed water.

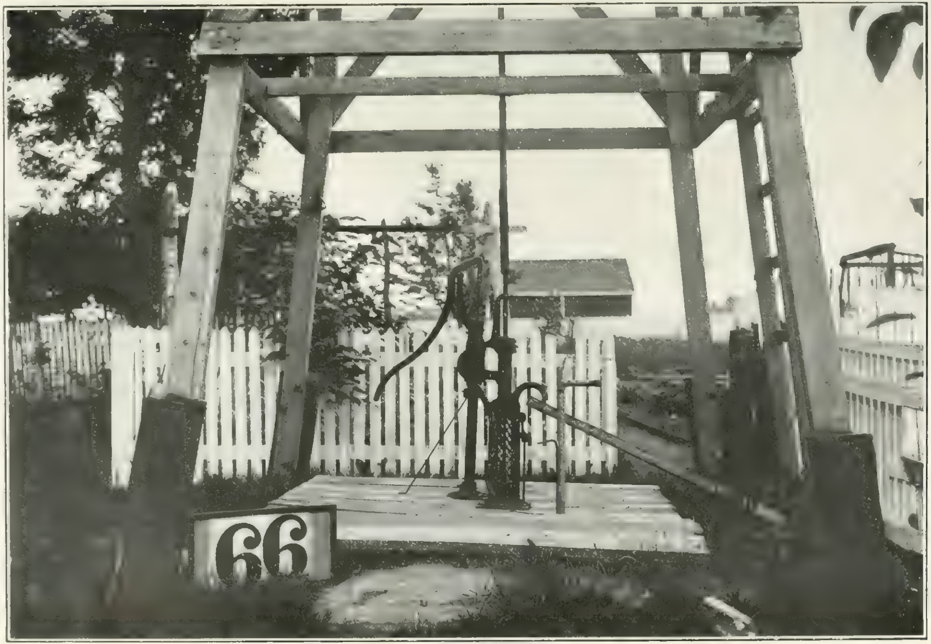
Photograph No. 58 shows an arrangement which seems to leave nothing to be desired. The well in this case is over 100 feet deep through rock, the barnyard lies off to the left and is at least 10 or 12 feet lower than the well. The dwelling is to the left and in front, and is still lower and farther away than the barn. The situation of the well is inside the building near the top of the hill, seen at the right of the picture. This building is the dairy and the floor is cemented, and when it was visited it was found to be scrupulously clean.

SPRINGS.

Much that has been said above in regard to wells applies equally to springs, but in addition to the danger of pollution from surface drainage and from seepage if the spring is open it is liable to pollution from the introduction of impurities in dipping the water out. This source of contamination may be guarded against by inclosing the spring in a concrete casing on all sides and providing a tight cover and a pipe inserted through one side to allow the water to run out. The cover should be removable, however, to permit of the cleaning out of the sand which always in time accumulates in the spring. Instead of the concrete casing a section of wide terra-cotta drain tiling set in cement over the point where the water wells up out of the ground may be used to good advantage. The tiling should be provided with a tight-fitting cover and a pipe to allow the water to run off. Either of these two arrangements obviate the danger of polluting the water from dipping unclean vessels into it. Some springs excellently protected by a coping on three sides and in other ways are made

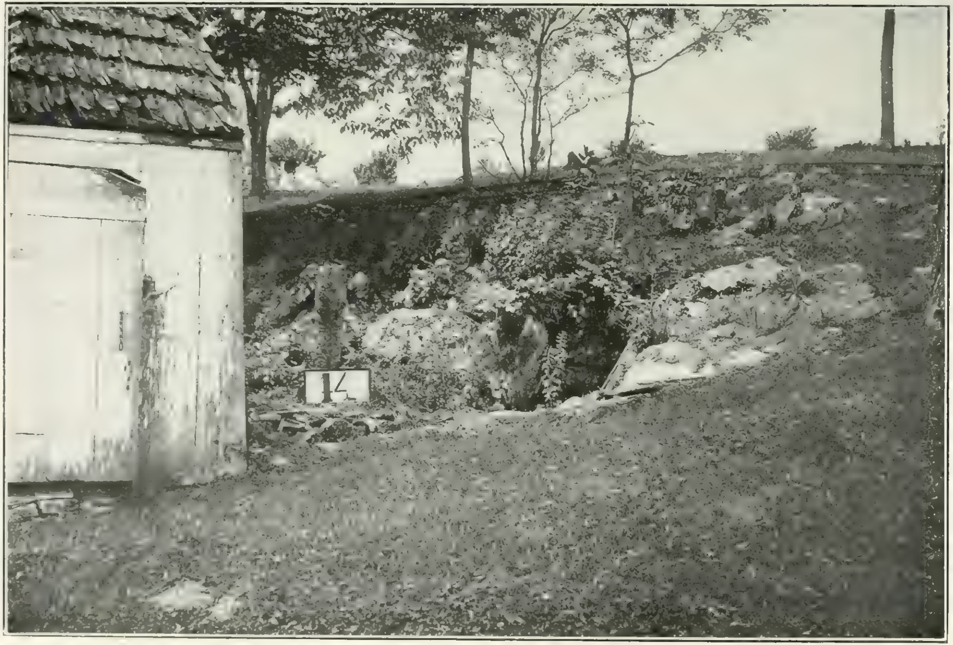


56. BAD PUMP SURROUNDINGS.

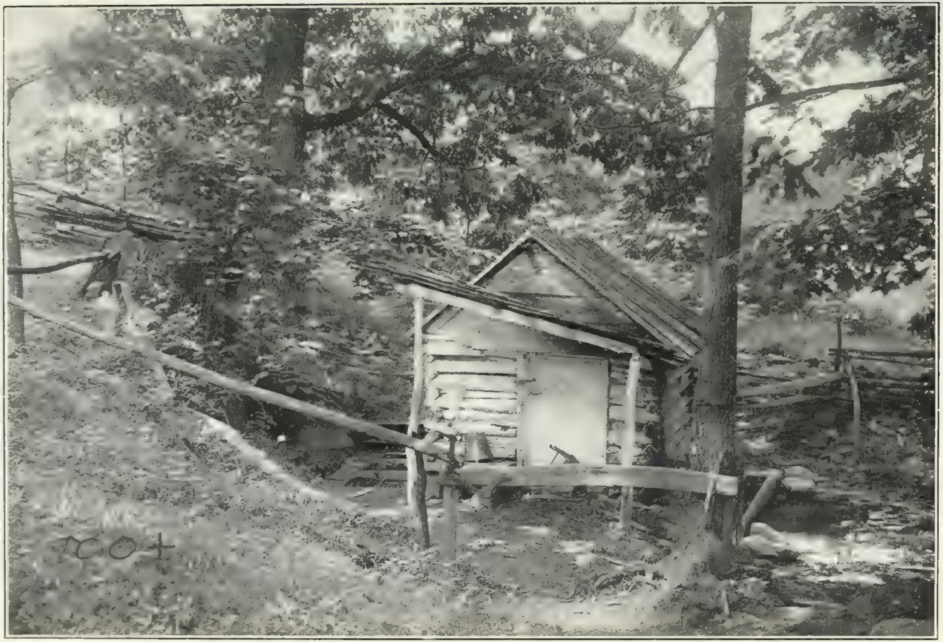


57. GOOD PUMP SURROUNDINGS.

58. GOOD WELL SITUATION IN BUILDING.



59. GOOD NATURAL SPRING SITUATION.



60. BAD NATURAL SPRING SITUATION.

liable to pollution by having steps leading down to the water's edge. Under such conditions the danger of introducing impurities from the soles of dirty shoes is of course apparent.

Unless a spring has a tight coping on all sides and is provided with a tight cover and spout, so that the water does not have to be dipped up, it falls short of the requirements of a sanitary supply. For convenience the water from the spring should be conducted by pipe to the house, dairy, and barn.

In the recent investigation but few springs were found which were properly protected. Some of them had cement or tight stone coping on three sides with steps leading down on the fourth side. Some had a tight coping on all sides, but the coping was level with the ground and the water thus exposed to pollution. But in many cases the water ran out of fissures in the rock into a natural or artificial basin, or bubbled up from the bottom of such a basin. In these springs no special care was exercised to guard against pollution.

The two photographs, numbers 59 and 60, given here show springs surrounded on three sides and over the top by natural rock but unprotected on the lower side in each case, and spring No. 44 is remote from sources of domestic contamination though it is in a pasture lot and is not fenced in. The dwelling is situated above and several hundred feet away up the hill shown to the right of the picture. The barn is still farther away in the same direction. The ground intervening between the house and the spring is in sod. The water is pumped up by a windmill to a tank near the house. The spring could be perfectly protected with little difficulty.

Spring No. 60 lies at the foot of the barnyard hill and is shown in the picture to the left of the dairy house. Some protection from wandering stock is afforded by the railing seen in the photograph. All the surroundings were untidy. There was filth up at the very edge of the spring. The fence seen to the left in the photograph incloses the hogpen, and above to the left is the barnyard, though it is true the slope does not incline directly from the barnyard to the spring. This is naturally an excellent supply and could be made to fulfill all sanitary requirements.

CISTERNS.

Where there is no spring and where it is not feasible for any reason to sink a well it becomes necessary to resort to cisterns, and if these are properly constructed and operated they may be made to fulfill all sanitary requirements. The walls should be water-tight, of course, both to prevent water from leaking out as well as to guard against pollution from without. The best cisterns are those constructed with two chambers divided by a porous brick partition through which the water is filtered. The water from the roof is made to run into one chamber and is pumped out of the other after passing through the partition. The rain pipe from the roof should be provided with an arrangement for preventing the first water which falls in time of rain from running into the cistern. The advantages of this arrangement are obvious, since the first water after dry weather may become polluted with dust or bird droppings on the roof. It will also serve as a cut-off after the cistern is filled. The water should be frequently pumped out and the cistern filled with fresh rain. The roof on which the rain is caught should be preferably of slate. Water from wooden shingles is often tainted.

ABUNDANCE.

The above considerations apply only to the purity of the water, but as stated in the beginning it is not sufficient for the demands of hygiene that the water should be pure, it should also be sufficient in amount for thorough cleanliness. The average amount of water used in various cities in America and in Europe by each inhabitant per day varies greatly, from 15 gallons in Vienna to 100 in Rome, 108 in New York, 120 in Detroit, 122 in Chicago, Ill. But this amount includes the water used for all purposes, manufactories, sprinkling, etc. A reasonable average amount for domestic purposes, as stated by Vernon Harcourt, is 25 gallons per day for each individual, and this is probably the amount which should be allowed on farms. Since the stock is usually watered at running streams this need not be taken into account in the reckoning. On farms generally the supply is ample. It may occasionally run short in times of prolonged drought, but there was no evidence of scarcity on any of the dairy farms recently visited.

CONVENIENCE.

Comparatively few farmers seem to realize the importance of convenience in the matter of water supplies even from a purely economic point of view, and much less from the bearing which such convenience has upon cleanliness and consequently upon health. Less than one-fifth of the dairy farms recently inspected have windmills, rams, or other mechanical means of bringing the water into the house or dairy.

Year after year on many farms water is pumped by hand or brought up the hill from the spring in buckets at the expenditure of a great amount of labor in the aggregate. Where it is at all feasible the water should be pumped into a tank and conducted at least into the dairy and the kitchen by pipe. Even where the water has to be pumped by hand it is desirable to have a tank for it insures abundance for purposes of cleanliness, but of course, if feasible, resort should be had to some mechanical device, windmill, engine, or ram for forcing water up to a tank to furnish a convenient supply for the house, barn, and dairy, in each of which there should be at least one spigot.

From the foregoing consideration it is evident that it is not usually a difficult matter to comply with all of the requirements of a sanitary water supply on the farm. It would appear to present much fewer difficulties than the same problem in towns, and seems to require only ordinary intelligence in selection of the site and subsequent management besides a certain expenditure of time and money necessary in the construction of devices for convenience. Each supply presents its own problem which must be solved for itself with proper recognition of the objects to be aimed at, and these are purity, abundance, and convenience.

18. METHODS AND RESULTS OF THE EXAMINATION OF WATER SUPPLIES OF DAIRIES SUPPLYING THE DISTRICT OF COLUMBIA.

METHODS AND RESULTS OF THE EXAMINATION OF WATER SUPPLIES OF DAIRIES SUPPLYING THE DISTRICT OF COLUMBIA.

By B. Meade Bolton, M. D.,
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The present investigation was undertaken in cooperation with the Dairy Division, Bureau of Animal Industry, Department of Agriculture, and had for its purpose the determination of the general sanitary condition by personal observation of the source of the supply, whether spring, well, or cistern; the location, surroundings, apparent liability of pollution due to proximity to the barnyard or domestic source of contamination or to careless and slovenly methods of drawing the water; furthermore in each case the purity of the water as indicated by the number of bacteria per 1 cubic centimeter and the presence or absence of the colon bacillus was determined by bacteriological examination of specimens plated on the spot and by chemical examination of samples sent as promptly as feasible after drawing. The chemical examination was made under the direction of Dr. T. M. Price, of the local laboratory in Washington. The counting of the plates to determine the number of bacteria and the culture tests for the detection of the colon bacillus were made by Dr. F. W. Tilley, of the Biochemic Division. Doctor Tilley has also assisted in the preparation of the accompanying tables. Dr. E. M. Santee, formerly of the dairy division, made photographs in most cases, and Mr. Clarence B. Lane, assistant chief of the Dairy Division, in others of the water supplies at the farms visited. These have all been included in a separate report to the chief of the Bureau of Animal Industry.

The investigation was started April 8, 1907, and was continued with some necessary interruption until September 11, 1907. It was made to include the dairies along the line of the Southern Railway from Vienna, Va., to Bluemont, Va., and consequently embraced those dairies which ship milk from Round Hill, Purcellville, Hamilton, Sterling, Herndon, Paeonian Springs, Clarkes Gap, Leesburg, Belmont Park, Ashburn, Wiehle, Hunter, Vienna, and also embraced most or all of the dairies around Frederick and Walkersville in Maryland. A bacteriological investigation had already been made and reported upon by Mr. Kellermann, Bureau of Plant Industry, of the dairies in the District of Columbia and in the country immediately contiguous thereto, and it was not deemed necessary to duplicate this work.

The method of procedure in the present investigation consisted in visiting from 4 to 8 or more dairies a day, and while the inspection of the dairies and barns was in progress, cultures were prepared from samples of the water, and the source and surroundings of the water supplies were recorded in each case upon a specially prepared blank form. Below is given a specimen of one of these forms which was actually employed.

In these blanks the parts applicable in each case were underscored, and the names, dates, and remarks filled in. In the accompaning blank the words which were underscored in this case are shown, and the parts filled in are shown in italics.

Name, *Buckingham Industrial School.* Date, *6–10–07.*
Address, *Buckeystown, Md.*

Covered open spring not protected from surface drainage by tight coping. Conducted through pipe by ram gravity.

Well bored. Open with bucket and windlass. Pump suction chain. Depth, *109* feet. *Through impervious layer rock.*

Cistern. Slate metal shingle roof. No means of turning off first water. Brick partition.

Situation *good* fair bad. Ground slopes *away* toward level. Ground *not* subject to pollution with human or animal excrement.

Remarks: *Well situated and well protected in all respects. Pump inside inclosed base of windmill tower. Door kept locked. Water conducted through pipe to dairy house about 100 yards away. All sources of pollution remote.*

[Front face filled out at the source of supply.]

Number of colonies per 1 c. c.:
At 35° C. for 24 hours,
At room temperature for 48 hours, 210.

PRESUMPTIVE TEST FOR B. COLI:

Number of acid-producing colonies on litmus agar per 1 c. c., *63.*
Neutral red agar:
Fermentation in one-tenth c. c.,— In 1 c. c.,+
Color in one-tenth c. c.,— In 1 c. c.,+

Milk *coagulated, reaction unchanged* Indol reaction *present.* Diagnosis *paracolon.*

[Reverse face filled out at the laboratory.]

In addition to the cultures, a half-gallon glass-stoppered bottle carefully cleaned in the laboratory was filled with the water under inspection, and shipped to Washington for chemical examination.

The media employed for obtaining the cultures consisted in all cases of litmus-lactose agar, and neutral-red-lactose agar. These media were both prepared without the addition of salt, and the reaction was brought to +1.5 American Public Health Association scale. The neutral-red-lactose agar contained 0.1 per cent of a 5 per cent aqueous solution of neutral red. The litmus-lactose agar was employed to make Petri dish plates. The neutral red-lactose agar was used for shake tubes. The amounts of water taken for the inoculation of the media were the same in each case. A sample of the water was caught in a sterilized test tube in each case and a melted tube of each of the media was inoculated by means of a graduated sterile pipette with 0.1 cubic centimeter of the water, and one tube each with 1 cubic centimeter. The litmus-lactose agar tubes were poured into the Petri dishes, and the neutral-red-lactose agar tubes were kept upright until the agar had set, after the water was thoroughly mixed with the medium. These plates and tubes were shipped to Washington as promptly as feasible, where they were taken in charge upon their arrival, and the neutral-red-lactose agar tubes were put at once into the incubator at 35° C. and left for forty-eight hours.

In order to protect the pipettes from accidental contamination each was wrapped separately in a piece of toilet paper. A special double capsule of thin wrapping paper was designed for the protection of the Petri dishes. This consisted of two cases, one slipping inside the other after the manner of a pocket cigar case. But these were abandoned for ordinary manila paper bags, which answer the purpose very well and are very cheap. The size and kind of bag employed were common 2-pound bags with the ends either folded over or cut off. The illustration of the traveling kit shows the pipettes and Petri dishes wrapped up as described.

In order to prevent the water, which condenses on the inside of the cover of the Petri dishes when the agar is poured, from condensing and running on the agar, each dish was covered with a disk of filter paper before sterilization. In a dish covered in this way the water condenses on the inside of the cover, but it is absorbed by the filter paper.

Various boxes were designed for carrying the necessary apparatus into the field, but the field kit which was found most satisfactory, and which was most used consists of a light wooden box, 14 inches high, 7½ inches deep and 5½ inches wide, outside measurements (Fig. 61). This box is divided into 2 sections of unequal depth hinged together. The shallower section is 2½ inches deep inside, and is divided into compartments for holding a square can of alcohol, and for test tubes of culture media and empty sterilized test tubes for collecting samples of water. The deeper section measures 13¾ inches in height, by 4½ inches in depth and 4½ inches in width, and is divided into compartments for Petri dishes and for an alcohol lamp and a tin can for use in melting the media. As will be seen in the figure, provision for holding the Petri dishes in the compartment consists of a vertical strip of wood on either side of the compartment. These strips do not extend up quite to the top, but stop 1 inch short, so as to allow for slipping the dishes in in a horizontal position while the agar is not yet firmly set. The compartment for the alcohol lamp below that for the Petri dishes needs no special description. Clips made of a clock spring serve to hold the pipettes, and these clips and the 2 cups for holding the ends of the pipettes at the bottom may be seen at each side of the compartments for the lamp and the Petri dishes. The compartments for the test tubes and extra supply of

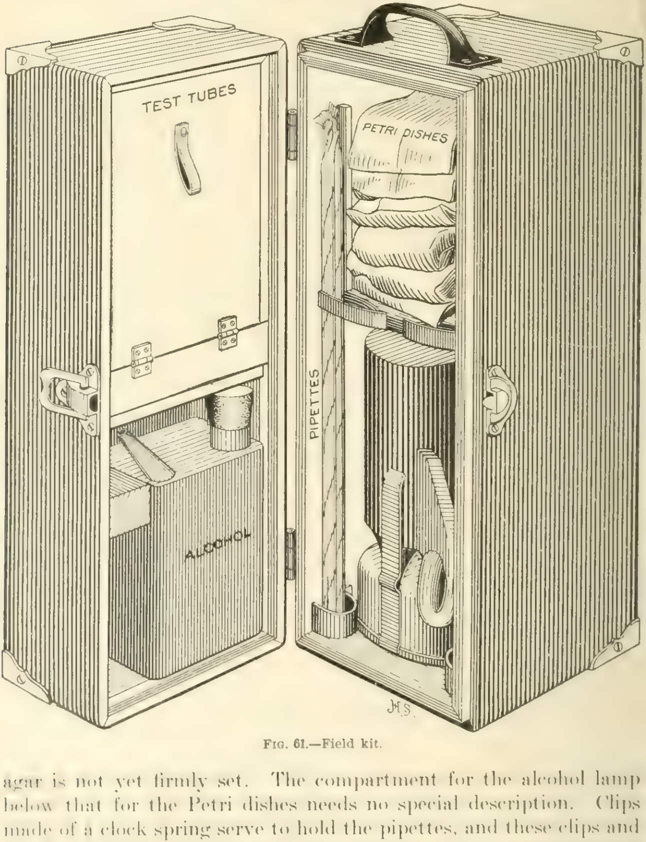

FIG. 61.—Field kit.

lcohol will be noticed in the illustration. The door which closes the ompartment for the test tubes is hinged so as to open downward, nd is kept from falling entirely open by a thin metal stop on either ide. The lamp (Fig.62) perhaps deserves a few words of descrip-ion. It is a vapor lamp of the kind which may be bought in any ardware shop. But it was fitted with a gauze cylinder resting on he burner, and a thin sheet-tin chimney, which add greatly to he heating power, and the tin cylinder also protects the flame from the wind. It also served the further purpose of a support for the vessel in which the culture tubes were melted. A deep, narrow tin cup with a wooden handle which just fit over the tin chimney when inverted was used to boil the water to melt the agar tubes.

In the kit just described it is possible to accommodate test tubes, Petri dishes, and pipettes sufficient for making tests from three or four places, provided the test tubes are moderately small in caliber and the Petri dishes not too deep. The sizes of those which were found convenient are test tubes without lips 15 millimeters in diameter by 150 millimeters long, and dishes 15 millimeters deep by 100 millimeters in diameter.

In addition to the above kit, 5 supply cases (Fig.63), also used for shipping, were employed. This case, as the accompanying illustration shows, consists of a box with a deep lid hinged on, or more properly of 2 boxes, one of which is shallower than the other, hinged together and closed by a catch. One of these compartments is fitted below for holding test tubes; the upper strip seen in the illustration can be removed for convenience in taking out the tubes and replacing them. The narrow compartment in the upper part is for pipettes. The other section is provided with compartments for Petri dishes, such as described in the field kit, and also a compartment for pipettes. This compartment was used for a supply of sterilized pipettes and the compartment for pipettes in the other section was used for the pipettes after they had been used. Both sections measure 14 inches from front to back and 10 inches from bottom to top. The shallower section is 3 inches deep and the deeper section 4½ inches deep. The compartments for the Petri dishes are 4½ inches wide. These are inside measurements. The case accommodates Petri dishes and test tubes for about 10 tests of the kind already described, each test requiring 2 tubes of neutral-red-lactose agar, 2 tubes of litmus-lactose agar, 1 sterile empty test tube, 1 pipette and 2 Petri dishes. One supply case and the field kit together hold sufficient apparatus for about 14 or 15 tests.

The field kit and one of the supply cases just described were taken along each day on the circuit of dairies visited and as soon as the

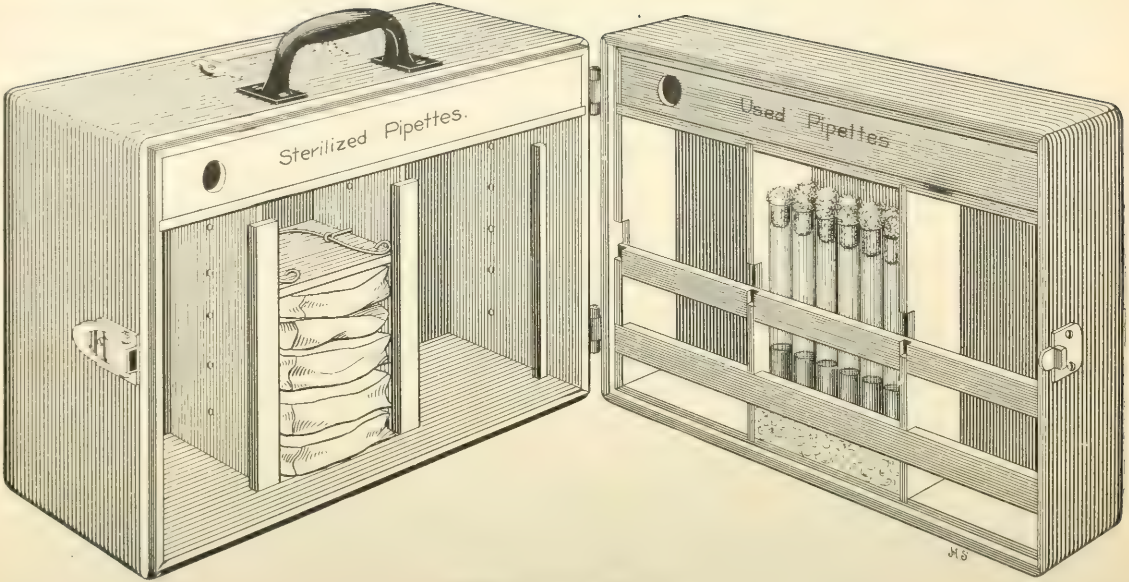

FIG. 62.—Supply case used for shipping.

lates and the shake cultures in neutral-red-lactose agar were made hey were transferred to the supply case and this was shipped to

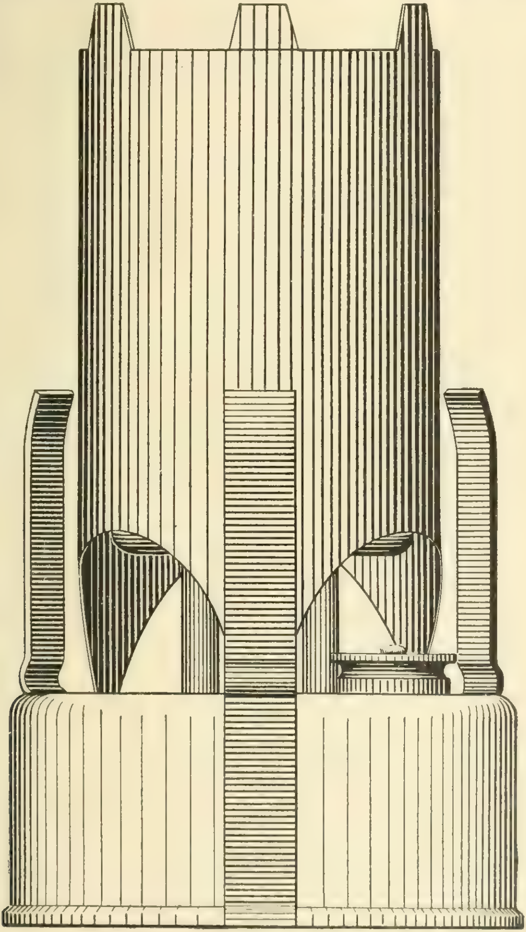

FIG. 63.—Alcohol vapor lamp with the tin cylinder in place.

Washington usually in the evening of the day on which the cultures were taken.

The cultures arrived in good condition except in a few cases, and in every case it was possible from one or both Petri dishes to make a determination of the number of bacteria per 1 cubic centimeter of the water.

It was found desirable to visit a certain number of dairies a second time after an interval of about four or five months from the date of the first visit, in order to check up and supplement the results obtained upon the first occasion.

The results of the investigation are given in detail in a report to the chief of the Bureau of Animal Industry which also includes descriptions of the places visited with photographs of many of them. In general the results show that there were 140 places visited in Maryland and 150 in Virginia. Of the Virginia dairies, 53 were visited a second time. At some of the dairies 2 different supplies were used, either 2 wells or a well and spring or either of these and a cistern. More wells than either springs or cisterns are used and more springs than cisterns. In Maryland there are 92 wells, 42 springs and 14 cisterns. In Virginia 75 wells, 63 springs and 17 cisterns.

In assigning a rating from the sanitary survey, account was taken of the general lay of the land roundabout, the liability to pollution from seepage, and the liability to pollution by direct introduction of impurities down the shaft in the case of wells, and into the basin in the case of open springs.

In pronouncing upon the presence or absence of pollution from the result of the chemical analysis, it has been necessary to assume some limits for purity, and from various statements given in the books and from the experience gained in the present investigation a maximum of 0.01 parts per million for nitrates, 0.02 for free ammonia and 0.05 of albuminoid ammonia were adopted as standards. The maximum allowed for chlorine varied of necessity for different localities. In the Maryland dairies the maximum found in the well water free from suspicion was 17 parts per 1,000,000, the minimum was 5.

In the Maryland springs otherwise free from suspicion the minimum amount of chlorine was 4 parts per 1,000,000, the maximum 6. In Virginia some of the wells which were free from suspicion showed as much as 63 parts per 1,000,000; while others showed only 5 parts per 1,000,000. Those in Virginia showing the larger amount lie in the region with Ashburn for a center and extending west to Leesburg and east to Herndon. In this district the minimum amount of chlorine was 11 parts per 1,000,000; the maximum 75. Outside of these limits the amount of chlorine averaged about 23 parts per 1,000,000 in the wells which showed no evidence of pollution as far as other criteria are concerned.

The minimum for the springs in Virginia showing otherwise no signs of pollution was 2 parts per 1,000,000; the maximum 20.

In regard to the total number of bacteria per 1 cubic centimeter it may be stated that it was not large as a rule and in many cases it ran low. In the water from wells in Maryland pronounced good on sanitary survey and which showed no evidence of pollution otherwise, the number ran from 10 to about 500 per cubic centimeter, except in 2 cases where the numbers were 1,580 and 2,900, respectively.

In the Maryland wells which were above suspicion the numbers ran from 15 to 320.

In the Virginia wells which were above suspicion the number of bacteria per 1 cubic centimeter ran from 0 to 510. In the Virginia springs which were above suspicion the numbers ran from 0 to 120, except in 1 case where the number ran up to 8,900.

The presence or absence of *B. coli* in the water of dairies is perhaps of special significance. By some the presence of this organism in water is interpreted to signify that the water is polluted with human feces and that if this is true typhoid feces may gain access. The presence of the *B. coli* is thus regarded by some as sufficient ground for condemning and closing the source of supply in such cases. It is true that there are others who are more reserved in their opinion of the significance of the *B. coli* in water and others again who are inclined to deny any such significance. It is not the purpose of the present paper to discuss the relative merits of any of these views, but to merely state what would appear to be the significance of the presence of *B. coli* in the water supply of the dairies under examination.

The occurrence of *B. coli* in some of the wells with apparently tight coping and cover, driven through rock, would seem difficult to account for. One case in particular seems remarkable. Well No. 24 of the Virginia dairies seems peculiarly well situated as regards likelihood of pollution of any kind, and 2 chemical examinations made about three months apart failed to show any indication of pollution. It is situated on the summit of a high hill several hundred yards from and 60 or 75 feet above the barnyard, and still farther and higher above any dwelling house. The surroundings are all clean. The ground is rock and the pump is placed on a high coping which is apparently tight. Yet the bacteriological examination showed high bacteria count on two examinations, too many in $\frac{1}{10}$ cubic centimeter to count in one case and 800 per 1 cubic centimeter in the other. *B. coli* was detected on both occasions. Other examples are not wanting in which similar results were obtained. The probability of pollution with human feces in all such cases seems very remote if not quite impossible. On the other hand it does not seem improbable that the cover of the well may have minute cracks between the boards, even where inspection fails to reveal any such defects, and that filth from the shoes may wash down through these cracks. It is to be assumed that this is the case with the well just described and other similar wells.

The presence of *B. coli* in open springs and cisterns may also be more readily explained by assuming its introduction through faulty manipulation than by pollution from seepage through the ground. In the case of open springs it seems indeed almost unavoidable that filth brought in on the shoes should be introduced into the spring. In bringing the milk from the cow barn to the dairy more or less cow dung is unavoidably tracked on the feet into the dairy and where there is an open spring it must be defiled from time to time. As cow dung as well as human feces contains *B. coli*, this would account for the frequent presence of the organism in the water from springs at dairies.

But whatever its source, *B. coli* should be specially guarded against in water used for washing vessels for containing milk. For aside from the fact that its presence may indicate fecal pollution, human or animal, it is likely to get into the milk, and when this occurs it multiplies rapidly and causes changes in the milk which render it unwholesome, particularly for children.

Still if the supposition stated above is correct that *B. coli* gains access to the water through careless methods in bringing filth on the shoes or otherwise, this can be readily remedied in many cases by the means recommended in the article on "Sanitary water supplies for dairy farms" in this same volume.

A careful examination should make it possible to decide the cases in which the water supplies can be guarded against pollution in the manner indicated and those cases where no remedy seems available. Even in cases where the chemical and bacteriological examinations fail to show pollution, it is sometimes apparent from a sanitary survey that a water supply is exposed to accidental pollution from carelessness and that this danger could be avoided by proper coping or otherwise. Dairies 103 and 119 in Maryland are examples in point. These dairies each obtain water from open springs and the location is rendered bad by the fact that in both cases the barnyard drains toward the spring and the surroundings in both cases are very slovenly. It would seem that pollution from animal feces at least would most certainly occur at frequent intervals. Yet the chemical and bacteriological examinations in these cases would indicate exceptionally pure water. The fact that the springs are both very bold and the supply of water is constantly being renewed in large quantiies probably accounts for the discrepancy. In both of these cases he supplies might be made to comply with sanitary laws.

In the following table is a synopsis of the results obtained in the present investigation.

The symbols used in the column "Sanitary rating" indicate, respectively, "G" for good, "B" for bad, and (?) for suspicious; in the column "Chemical analysis," + for more or less evidence of pollution according to the standards given − for cases in which the substances indicative of pollution were found to be within limit prescribed for unpolluted water; under *B. coli communis*, + for the presence of the organism, − for its absence. Thus in the first line it is seen that there were 3 of the wells and 3 of the cisterns in Maryland, and 4 of the wells and 4 of the cisterns in Virginia which were apparently good as far as sanitary inspection showed, but which were polluted according to the chemical analysis and also contained *B. coli*, while all the springs which are classified as "G" in the table do not all come fully up to sanitary requirements, it was considered advisable to classify those as such which have no obvious sources of pollution within a short distance and which have more or less provision against contamination by way of coping. In reality there are very few springs which come up to the requirements stated in the article on "Sanitary water supplies for Dairy farms" in the present volume.

TABLE I.—*Maryland dairies.*

Sanitary rating.	Chemical analysis.	B. coli communis.	Number of wells.	Number of springs.	Number of cisterns.
G	+	+	4	......	3
G	+		6	......	4
G		+	8	2	2
G			22	10	......
G	?	+	12	1	1
G	?	−	8	4	1
B	+	+	3	2	......
B	+		3	1	......
B		+	3	3	......
B			3	3	......
B	?	+	1	6	1
B	?		1	1	1
?	+	+	3		...
?	+		1	3	...
?		+	2	1	......
?			1	3	.
?	?	+	7	1	1
?	?	−	4	1	......

The salient points shown by the above table are that of 60 wells in Maryland which were pronounced good on sanitary inspection, 1 showed chemical and bacterial pollution, 6 showed chemical pollution only, 8 showed bacterial contamination only, 22 showed neither, 20 were suspicious chemically, and of these 20 there were 12 which showed the presence of *B. coli.*

There were 36 which failed to show the presence of *B. coli* and 30 which did not show chemical pollution.

Of the 17 springs in Maryland pronounced good on sanitary inspection, 2 showed bacterial pollution alone, 10 were found to be unpolluted either chemically or bacteriologically, 5 were suspicious chemically, and one of these showed the presence of *B. coli.* In all, 14 did not show *B. coli*, 10 did not show chemical pollution, and 5 showed some evidence of chemical pollution.

Of the 14 wells in Maryland which were pronounced bad on sanitary inspection, 6 showed evidence of chemical pollution, 6 showed no evidence of such pollution, and 2 were suspicious. Three showed both chemical and bacteriological pollution and 3 showed neither.

Of the 19 springs classed as bad from the sanitary survey, 6 showed evidence of chemical pollution, 6 showed no evidence of chemical pollution, and 3 showed neither chemical nor bacterial pollution. Seven were suspicious from chemical analysis, and 6 of these contained *B. coli.*

Of 9 springs which were classed as suspicious from the sanitary survey, 3 showed neither chemical nor bacteriological pollution. None showed both.

All the cisterns showed either bacterial or chemical pollution or both.

TABLE II.—*Virginia dairies.*

Sanitary rating.	Chemical analysis.	B. coli communis.	Number of wells.	Number of springs.	Number of cisterns.
G	+	+	4		4
G	+			1	1
G		+	7	10	.
G			9	8	1
G	?	+	8	3	4
G	?		3	.	......
B	+	+	.	1	......
B	+	—		1	.
B		+	2	6	
B		—		5	
B	?	+	1		
B	?		2	3	
?	+	+	1		3
?		+	3	1	2
?	—	—	2	1	..
?	?	+	2	2	2
?	?		......	1	......

Table II shows the Virginia dairies visited only once. Of the 31 wells of this series which were pronounced good on sanitary inspection, 9 showed neither chemical nor bacteriological pollution, 4 showed both, 19 showed *B. coli*, 16 showed chemical pollution.

In the springs visited once, 22 were pronounced good on sanitary inspection, and 8 of these showed neither chemical nor bacteriological pollution, 18 of these showed no chemical pollution, 9 no bacteriological pollution, 1 showed distinct chemical pollution, and 13 bacteriological pollution.

Of the 16 springs which were pronounced bad from the sanitary survey, 5 showed neither chemical nor bacteriological pollution, 11 showed no chemical pollution, 9 showed no bacteriological pollution.

Only 1 of the 17 cisterns showed neither chemical nor bacteriological pollution, 7 showed both, and the others showed either one or the other form of pollution.

TABLE III.—*Virginia dairies examined twice.*

Sanitary rating.	Chemical analysis.	B. coli communis.	Wells.	Springs.
G	+	+		1
	+	+		
G	+	+	1	1
		+		
G		+	2	2
		+		
G			1	..
	+	+		
G	−	+	6	6
G			1	1
G	[illegible]	+		1
	[illegible]	+		
G	[illegible]	+	2	
G	+		1	
	+			
G	[illegible]	+	2	
	[illegible]			
G		+	1	2
	[illegible]			
B	+	+	3	
	+	+		
B		+		3
		+		
B				1
	[illegible]	+		
B	?			1
	−	+		
?	[illegible]	+	[illegible]	
	?			

TABLE III.—*Virginia dairies examined twice*—Continued.

Sanitaay rating.	Chemical analysis.	B. coli communis.	Wells.	Springs.
?			.	1
?	+	+	2	....
		+		
?	+	+	1	..
		+		
?	+	+	2	....
		+		
?		+	1	....
		+		
?	+	+	1	......
	+			
?		+	1	....
?		+	.	1

Table III shows the Virginia dairies which were visited twice. Of the 31 wells of this series, 17 were rated good on sanitary survey, 3 were rated as bad, 11 as suspicious. Only 1 of those rated as good failed to show either chemical or bacteriological pollution. The 3 rated as bad all showed both chemical and bacteriological pollution.

Of the 21 springs visited twice 14 were rated as good from the sanitary survey, 5 were rated as bad, 2 as suspicious. One was found unpolluted chemically or bacteriologically on both examinations, and the others were found more or less polluted either chemically or bacteriologically on one or the other visit.

The above analysis of the results seems to show that there are comparatively few water supplies on the dairy farms which were visited which are free from sanitary objection, but in spite of this fact it is nevertheless probable that in many or most cases the faults can be rectified. In fact the faults have already been corrected in some cases where they were pointed out to the owners of the dairies. It would seem advisible in some cases to close up the source of supply, but in most cases all that would seem necessary is to point out to the dairymen the sources of pollution, and to give them instructions in regard to their avoidance.

19. THE CLASSIFICATION OF MARKET MILK.

THE CLASSIFICATION OF MARKET MILK.

By A. D. MELVIN,

Chief of the Bureau of Animal Industry, United States Department of Agriculture.

In providing for the classification of milk, the primary object is to exclude all milk which may be harmful to the consumer and to provide for milk that will be wholesome and nutritious. In doing so the restrictions should not be more burdensome than is necessary to accomplish this result, and should not be so severe as to lessen materially the quantity and so eliminate milk, on account of increased price, as an article of diet for the poor. In view of the danger of using raw milk from diseased cows, drawn in improperly constructed dairies and under insanitary conditions, and improperly handled, it is imperative that no raw milk be sold to the public except when those in authority know that it is safe to be used in that condition.

Raw milk of the highest standard should be produced for the use of infants and invalids and for those who desire to use only such milk and who are willing to pay for the greater cost of production. A wholesome supply of raw milk which can be furnished without involving unreasonable cost should also be provided for others. A third class includes undoubtedly the largest proportion of milk which is at the present time furnished for consumption and is unsafe for consumption in the raw state. To remove immediately from consumption this latter supply would result in such an increase in the cost of all milk as to place milk as an article of diet beyond the reach of the poor. It seems necessary, therefore, to provide some means of rendering safe all milk of doubtful quality, and this can be done under present conditions only by pasteurization under proper supervision.

The tuberculin test when applied by the Bureau of Animal Industry during the period from April 1, 1907, to June 30, 1909, to 2,471 cows supplying milk to the city of Washington showed that 15.25 per cent of those tested were affected with tuberculosis, and it is believed that just as high a proportion of diseased cows will be found in the dairy herds supplying many other cities. These cows should be replaced by healthy ones. The work of testing with tuberculin all cows supplying milk to a city of considerable size is an immense undertaking and must necessarily consume much time. Much time is required also in providing suitable cow stables, milk houses, and a pure water supply in all instances where such essential features are lacking. Facilities must also be provided for delivering milk at a reasonable temperature.

It has been demonstrated that cows showing no physical evidences of tuberculosis may be affected with tuberculosis and secrete milk containing germs of that disease; also that the feces of such cattle are contaminated with that bacillus, and particles of such feces can readily gain entrance into milk. It seems important, therefore, in any classification of milk for city use, to require pasteurization of all doubtful milk for some time, or until all doubt as to the wholesomeness of the milk is removed.

It is therefore recommended that milk be graded in three classes, (1) certified milk, (2) inspected milk, and (3) pasteurized milk, in accordance with the specifications following, and that this classification be embodied in laws and regulations and enforced by public health authorities.

CLASS 1.—CERTIFIED MILK.

The use of this term should be limited to milk produced at dairies subjected to periodic inspection and the products of which are subjected to frequent analyses. The cows producing such milk must be properly fed and watered, free from tuberculosis, as shown by the tuberculin test and physical examination by a qualified veterinarian, and from all other communicable diseases, and from all diseases and conditions whatsoever likely to deteriorate the milk. They must be housed in clean, properly ventilated stables of sanitary construction, and must be kept clean. All persons who come in contact with the milk must exercise scrupulous cleanliness and must not harbor the germs of typhoid fever, tuberculosis, diphtheria, or other infections liable to be conveyed by the milk. Milk must be drawn under all precautions necessary to avoid infection, and be immediately strained and cooled, packed in sterilized bottles, and kept at a temperature not exceeding 50° F. until delivered to the consumer. Pure water, as determined by chemical and bacteriological examination, is to be provided for use throughout the dairy farm and dairy. Certified milk should not contain more than 10,000 bacteria per cubic centimeter, and should not be more than twelve hours old when delivered. Such milk should be certified by public health officers or by some other properly constituted authority.

CLASS 2.—INSPECTED MILK.

This term should be limited to clean raw milk from healthy cows, as determined by the tuberculin test and physical examination by a qualified veterinarian. The cows are to be fed, watered, housed, and milked under good conditions, but not necessarily equal to the conditions prescribed for class 1. All persons who come in contact with the milk must exercise scrupulous cleanliness and must not harbor the germs of typhoid fever, tuberculosis, diphtheria, or other infections liable to be conveyed by the milk. This milk is to be delivered in sterilized containers and is to be kept at a temperature not exceeding 50° F. until it reaches the consumer. It should contain not more than 100,000 bacteria per cubic centimeter.

CLASS 3.—PASTEURIZED MILK.

Milk from dairies which do not comply with the requirements specified for classes 1 and 2 should be pasteurized before being sold, and should be sold under the designation "pasteurized milk." Milk for pasteurization should be kept at all times at a temperature not exceeding 60° F. while in transit from the dairy farm to the pasteurizing plant, and milk after pasteurization should be placed in sterilized containers and delivered to the consumer at a temperature not exceeding 50° F.

All milk of unknown origin should be placed in class 3 and subjected to clarification and pasteurization. No cow in any way unfit for the production of milk for use by man, as determined upon physical examination by an authorized veterinarian, and no cow suffering from a communicable disease should be permitted to remain on any dairy farm on which milk of class 3 is produced, except that cows which upon physical examination do not show physical signs of tuberculosis may be included in dairy herds supplying milk of this class.

This milk is to be clarified and pasteurized at central pasteurizing plants, which should be under the personal supervision of an officer or officers of the health department. These pasteurizing plants may be provided either by private enterprise or by the municipality, and should be located within the city.

By the term "pasteurization" as used herein is meant the heating of milk to a temperature of 150° F. or 65° C. for twenty minutes, or 160° F. or 70° C. for ten minutes, as soon as practicable after milking, in inclosed vessels, preferably the final containers, and after such heating immediate cooling to a temperature not exceeding 50° F. or 10° C.

OTHER CONDITIONS.

No milk should be regarded as pure and wholesome which, after standing for two hours or less, reveals a visible sediment at the bottom of the bottle.

No dairy farm should be permitted to supply milk of a higher class than that for which its permit has been issued, and each dairy farm supplying milk of a specified class should be separate and distinct from any dairy farm of a different class. The same owner, however, may supply different classes of milk, providing the dairy farms are separate and distinct.

The term "milk" as herein used includes cream.

20. CERTIFIED MILK AND INFANTS' MILK DEPOTS.

CERTIFIED MILK AND INFANTS' MILK DEPOTS.

By JOHN W. KERR,
Assistant Surgeon-General, Public Health and Marine-Hospital Service.

"She can milk; look you, a sweet virtue in a maid with clean hands." —*Shakespeare.*

The increasing complexity of community life with its attendant evils has had an influence in the reduction of maternal feeding of infants and at the same time has rendered less accessible a supply of wholesome artificial food.

Educational measures are therefore demanded for the restoration of the function of the female breast. In the meantime a pure supply of cow's milk for clinical purposes is of vital importance, a fact becoming more and more recognized by physicians and others interested in the reduction of infant mortality and the improvement of conditions among the poor.

Its importance is also emphasized by sanitarians, who have reported no less than 500 epidemics of typhoid fever, diphtheria, and scarlet fever within the last half century in which the infection was transmitted by infected milk.

In consequence of a just appreciation of these conditions measures have been adopted in various sections of this and other countries to prevent the enormous waste of human life which is known to occur within the first year after birth—due mainly to a lack of proper food.

Through private initiative two notable movements were started in the United States in 1889 and 1893, respectively; the first had for its object the control and distribution of milk to infants of the poor and the education of mothers in infant hygiene; the second, the production under the control of a medical milk commission of pure or "certified" milk for clinical purposes.

CERTIFIED MILK.

The term "certified milk" was coined by Dr. Henry L. Coit, of Newark, N. J., who in 1892 formulated a plan for the production of pure milk under the auspices of medical milk commissions. This plan contained the following general requirements:

> First. That physicians give their practical support to an effort conducted by a medical milk commission selected by a medical society which shall endeavor to bring to the city a supply of milk produced under such regulations that purity shall be assured.
>
> Second. That approved and trustworthy dairymen possessing honor, financial ability, and dairy facilities shall be induced by reason of promised medical support and the increased price of their milk to conduct their dairies, collect, and handle the product in conformity with the code of requirements made by the aforesaid medical commission and imposed by it in due legal form.
>
> Third. That the duties of the commission shall be, first, to establish correct clinical standards of purity for cows' milk; second, be responsible for a periodical and personal inspection of the dairy or dairies under its patronage; third, to provide for bimonthly expert examination of the dairy stock by competent and approved veterinarians and for medical supervision of the employees by competent physicians.
>
> The milk produced shall also be subject to periodical chemical analysis and to bacterial counts made under the direction of the commission as often as in its judgment is desirable. The experts employed by the commission shall render their reports to this body, which constitute the basis of its certification of the product.
>
> The expense of examinations and inspections shall be defrayed by the dairymen; but the members of the commission shall receive no pay for their services
>
> The findings of the commission shall be published to the profession only, and the milk thus produced shall be known as "certified milk" and be sold in quart containers bearing the date of milking and the seal of the commission.[a]

In 1893 the Medical Society of Essex County, N. J., adopted this plan and organized the first medical milk commission in the United States.

A dairyman was found who was willing to undertake the production of milk according to the following standards of purity formulated by Doctor Coit in connection with the original plan:

> First. An absence of large numbers of micro-organisms and the entire freedom of the milk from pathogenic varieties.
>
> Second. Unvarying resistance to early fermentative changes in the milk, so that it may be kept under ordinary conditions without extraordinary care.
>
> Third. A constant nutritive value of known chemical composition and a uniform relation between the percentage constituents of fat, proteid, and carbohydrate.

The following formal contract was therefore signed May 19, 1893, under which periodical inspections of the dairy, veterinary examinations of the herd, chemical analyses and bacteriological counts of the milk were instituted:

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[a] Coit, H. L. Brief history of the development of the pure milk movement in the United States.

Copy of the Agreement Between the Medical Milk Commission of Essex County, N. J., and Stephen Francisco, of Caldwell, N. J., Dated May 19, 1893.

The following agreement, made this 19th day of May, 1893, between Henry L. Coit, M. D., of Newark, N. J., Theron Y. Sutphen, M. D., of Newark, N. J., William B. Graves, M. D., of East Orange, N. J., L. Eugene Hollister, M. D., of Newark, N. J., Joseph W. Stickler, M. D., of Orange, N. J., and James S. Brown, M. D., of Montclair, N. J., parties of the first part, and Stephen Francisco, of Caldwell, N. J., party of the second part: Witnesseth as follows, that the party of the second part doth hereby bind himself to a fulfillment of the provisions of this contract for and in consideration of the benefits hereinafter named by the parties of the first part.

Furthermore, the following-named persons, Frank A. Wilkinson, of Newark, N. J., Isaac Lane, of Caldwell, N. J., and William Bush, of Caldwell, N. J., all acquaintances of the party of the second part, hereby affix their signatures to this agreement, attest to the honor of the party of the second part, and become sureties for the execution of this agreement.

1. The party of the second part doth hereby agree to conduct such parts of his dairy as may be hereinafter named, collect and handle its products in conformity with the following code of requirements, for and in consideration of the promised indorsement of the parties of the first part, as hereinafter indicated. The milk thus produced shall be known as certified milk, shall be designed especially for clinical purposes, and when at any time the demand shall be greater than the supply and is required by a physician, either for infant feeding or the diet of the sick, it is hereby agreed that such shall be the preferred purchaser.

2. The party of the second part further agrees to pay for chemical and bacteriological examinations of the aforesaid certified milk at such times as in the judgment of the parties of the first part is desirable.

3. He also agrees to defray the cost of a bimonthly inspection of his dairy stock or oftener, if necessary, by a competent and approved veterinarian, all of which persons, namely, the chemist, the bacteriologist, and the veterinary surgeon, shall be chosen by the parties of the first part, to whom they shall render their reports in writing.

4. It is expressly understood and agreed that the party of the second part shall not pay more than the sum of $500 in any one year for the services of chemist, bacteriologist, and veterinary surgeon, and the party of the first part shall limit the expense of such service to that amount. It is furthermore agreed that the party of the second part, on receipt of a certified copy of the reports of the experts, shall mail to the persons indicated by the parties of the first part, and not to others, a duplicate printed copy of the aforesaid reports bearing the signatures of the experts and the names of the physicians, the same to be issued at such intervals as in the judgment of the parties of the first part is desirable; also that the necessary expenditures for printing and circulation be met in the same way as herein provided for expert examinations.

LOCATION OF LANDS.

5. It is hereby understood and agreed that the lands used by the owners, agents, or assigns of the dairy conducted by the party of the second part and employed for pasturage, or any lands that may be hereafter acquired for such purpose, or such lands as may be used for the cultivation of hay or fodder, shall be subject to the approval of the parties of the first part.

BUILDINGS.

6. It is also understood and agreed that the buildings, such as stables, creamery, dairy house, and spring house, shall be constructed after the most approved style of architecture, in so far as construction may affect the health of the dairy stock or the character and conditions of the milk.

7. That the buildings used for the housing of the animals shall be situated on elevated grounds and capable of being properly drained.

8. Said buildings to be sheltered from cold winds, lighted, and ventilated according to approved hygienic methods. The buildings shall be constructed so as to favor the prompt and easy removal of waste products.

9. The apartments used for the storage of either feed or fodder shall be removed from possible contamination by stable waste or animal odors.

10. All buildings shall, in addition to healthful location, approved construction, and proper ventilation, be kept free from animal or vegetable matter in a state or process of decomposition or decay and always free from accumulations of dust or mold.

THE WATER SUPPLY.

11. The dairy shall be supplied with an abundance of pure water.

12. No water from shallow wells or springs holding surface drainage shall be used for watering stock, cooling milk, or cleaning vessels.

13. Nor shall any well or spring be located within 300 feet of the stable.

SURROUNDINGS.

14. It is further understood and agreed that the immediate surroundings of the buildings shall be kept in a condition of cleanliness and order. There shall not be allowed to accumulate in the vicinity any loose dirt, rubbish, or decayed vegetable or animal matter, or animal waste.

15. Nor shall there be within 300 yards of any building any constantly wet or marshy ground or stagnant pools of water.

16. Nor shall there be kept within 300 yards of any building used for dairy purposes any fowl, hogs, horses, or other live stock.

17. It is hereby understood and agreed that the following unhealthful conditions shall be a sufficient reason to exclude any animal from the herd used for any purpose in the aforesaid dairy. Any animal that is judged by a competent observer to suffer from tuberculosis, even though the disease be localized or latent.

18. Any animal with fever. Any animal suffering from septic absorption or other disease, followed or associated with parturition.

19. Any animal suffering from mammitis or mammary abscess.

20. Any animal with persistent diarrhea or any other abnormal physical condition which could in any way be detrimental to the character of the milk.

21. It is furthermore agreed that when an animal shall be found by a competent observer to be in a state of ill health, prejudicial either to the other animals in the herd or to human health, the same shall be removed immediately and, if necessary, shall be killed.

22 It is also understood and agreed that the party of the second part shall exclude from the herd used for producing certified milk, immediately after discovery, any animal subject to the following conditions: Any animal that was bred through consanguinity within a period of three generations.

23. And from this time forth any animal of those bred by the party of the second part used for producing certified milk that was not as a heifer kept sterile during its first 27 months.

24. Any phenomenal milker, except that glandular disease or tuberculosis has first been excluded by a competent observer.

25. It is furthermore agreed that if at any time it is desired by the parties of the first part that a different breed of milch cows should be substituted for the one in use, in order that the standards of quality in the milk may be raised, the party of the second part will endeavor to carry the same into effect.

HOUSING AND CARE.

26. It is furthermore agreed that the dairy stock employed in the production of certified milk shall be properly sheltered from the influences of weather and climate prejudicial to their health, also that the animals shall be kept clean, groomed every day, and treated kindly at all times.

27. The waste products of the stable shall be removed so frequently, and the stable floor so thoroughly cleaned, that the same shall be as free as possible from animal odors.

28. It is also agreed that no milch cow shall be used for dairy purposes while in a state of excitement, either as a result or during the period of estrux, or which has been made nervous either by beating, whipping, kicking, prodding, or running.

FEEDING.

29. It is hereby understood and agreed that the methods of feeding the cows furnishing the certified milk shall be subject to the approval of the parties of the first part. The feed and fodder shall consist only of nutritious and wholesome materials, such as grass, clover and timothy hay, whole grain, or the entire result of the grist. No materials shall be employed which are or may become injurious to the health of the animals. There shall not be fed at any time or in any quantity, either alone or mixed with other feed or fodder, hulls, screenings, wet or dry brewer's grains, sour ensilage, or any waste by-product in the treatment of grain, low marsh grass, or any of the questionable or exhausted feeds or fodders employed either to increase the milking capacity of the animal or that will produce an impoverished milk or that will impart to it unnatural odors or flavors. Nor shall the cows be allowed to eat green or worm-eaten fruit, poisonous weeds, or to drink poisonous or stagnant water.

COLLECTING AND HANDLING.

30. It is furthermore understood and agreed that the cows from which is obtained certified milk shall be milked only in a clean building and not in an illy ventilated stable containing foul odors and bad air.

31. No animal furnishing certified milk shall be milked until the udder shall first have been cleaned in a manner approved by the parties of the first part.

32. No person shall be allowed to draw the milk who has not within fifteen minutes of the milking first washed his or her hands, using soap and nail brush, and afterwards thoroughly rinsing the hands in clean water.

33. The person or persons engaged in milking shall also be dressed in clean overclothes.

34. No person shall be allowed to draw the milk who has been engaged with the care of horses in the same clothing or without first washing his hands.

35. No milk shall be represented as certified milk that is not received from the udder into vessels and from these into cooling cans, both of which are perfectly clean and dry, having been cleansed and heated at a temperature adequate to effect complete sterilization since the last milking, and have been kept inverted in a clean, dry, and odorless atmosphere.

36. No milk shall be represented as certified milk that has not been passed through a sieve of wire or other cloth, either while milking or immediately thereafter, having not less than 100 meshes to the linear inch.

37. No milk shall be represented as certified milk that does not consist of the entire contents of the udder at each milking, including the fore milk, middlings, and strippings.

38. No milk shall be represented as certified milk that has been drawn from the animal at abnormal hours, such as midnight or noon, nor from any animal for a period of

nine weeks before calving, or that has not been separated for nine days after parturition.

39. No milk shall be represented as certified milk which has been exposed to the emanation or infection of any form of communicable disease, either in the person or persons handling the milk or by accidental contamination in cleaning milk containers or by the association of any person engaged in handling the milk with person or persons sick of contagious disease.

PREPARATION FOR SHIPMENT.

40. It is hereby understood and agreed that all milk represented as certified milk shall receive every known detail of care that will promote its keeping qualities and favor its safe transportation.

41. That the milk on being drawn from the cow shall be treated by ice or clean, cold water in motion, and proper aeration, in order, first, to remove its animal heat, and second, to reduce its temperature to a point not above 50° nor below 40° F., said temperature to be acquired within forty-five minutes after milking and maintained within the above limits while held for shipment, during its transportation, and until it is delivered to the purchaser.

42. That the cooling of the milk shall not be conducted in the same building in which it is drawn, nor in an atmosphere containing dust or tainted with animal odors.

43. That all the foregoing provisions concerning the cleansing and condition of vessels or utensils shall be complied with in the said cooling process.

44. It is furthermore agreed that no milk shall be represented as certified milk that has been changed or reduced in any way by the addition of water or any solid or liquid substance, in or out of solution, or the subtraction or removal, in any manner, of any part thereof.

45. It is hereby understood and agreed that all milk to be represented as certified milk shall be packed in flint glass quart jars immediately after it is cooled.

46. Said jars to be of pattern approved by the parties of the first part.

47. It is furthermore agreed that the bottles or jars, before being used, shall be cleaned by hand, separately, with the aid of hot water, alkaline soaps, rotating brush, and steam, and that they shall be rinsed in two separate baths of clean, hot water and then thoroughly dried and kept inverted until used, without covers, in a clean, dry atmosphere free from odors.

48. It is agreed that the jars shall be filled by a method approved by the parties of the first part.

49. That they shall be sealed after all air has been excluded by the most approved device for closing them.

50. The bottles after being filled shall be labeled across the cap, bearing the words "Certified Milk," with the name of the dairyman, together with the date of milking.

51. It is furthermore agreed, that no milk shall be sold as certified milk that is more than three hours old when bottled nor more than twenty-four hours old when delivered.

TRANSPORTATION AND DELIVERY.

52. It is hereby understood and agreed that the transportation and distribution of all milk represented as certified milk shall be conducted by the party of the second part, either in person or by persons employed by him.

53. That in transit the milk shall not be exposed to any of the foregoing prohibitory conditions.

54. That it shall not be subjected to agitation.

55. That it shall not be exposed to the heat of the sun.

56. That the delivery wagons shall be so constructed that the required temperature of the milk may be maintained during transit.

57. That before the wagons are filled for shipment the body, the trays, and compartments shall be flushed with boiling water.

58. It is furthermore agreed that the distributing agents shall, during the transfer of the milk from the dairy to the purchaser, be subject to the following restrictions, namely, that they shall use no tobacco.

59. That they shall take no intoxicating drinks.

60. That they shall not collect the empty containers nor receive money or milk checks from houses in which an infectious or contagious disease is known to exist

61. It is also hereby agreed that the collection of empty bottles from places where infectious or contagious disease is known to exist shall be made by other persons than those employed to deliver the milk.

62. That these collections be made with wagons not employed in the distribution of the milk.

63. That before these empty bottles shall be returned to the dairy they shall be carried to a separate building and first be subjected to the process of cleaning bottles indicated in a former clause of this contract.

64. It is hereby understood and agreed that if any further precautions or changes in method calculated to improve the quality of milk or guard the same from impurities or dangers is desired that the party of the second part will cheerfully be governed by such additional rules and regulations as may be laid down by the parties of the first part.

65. It is understood and agreed by the party of the second part, the same binding the owners, agents, or assigns of the aforesaid dairy, that the product known as certified milk shall be under the following restrictions in its sale, namely, that until the amount required within the boundaries of Essex County shall first be supplied it shall not be sold beyond these limits except that the parties of the first part shall give their consent.

66. It is furthermore agreed by the party of the second part, the same binding the owners, agents, or assigns of the aforesaid dairy, that in the event of a failure to comply with any or all of the requirements of the foregoing contract that party of the first part shall reserve the right to withdraw from the contract and publish the fact in such manner as they deem best.

67. Finally, it is understood and agreed that nothing in this contract shall prevent the abrogation of any of the provisions of the same by the parties of the first part, provided that it shall be done for the purpose of substituting other provisions designed to promote the objects of their organization.

68. It is further understood and agreed by and between the parties hereto that the party of the second part shall be at liberty to cancel this agreement by giving two months' notice in writing of his desire to do so, in case of inability for any reason to comply with the terms of the same.

In witness whereof the said parties have hereunto set their hands, the day and year first above written.

At the suggestion of the commission the term "certified milk" was copyrighted by the Fairfield Dairy Company (the first to produce "certified milk"), the object being to prevent its use by any except medical milk commissions organized for the improvement of dairy hygiene.

The precedent established sixteen years ago has since been followed in many cities of the country, and no less than 56 commissions have been organized to encourage the production of pure milk for clinical purposes. The plan adopted by these commissions was practically the same as that formulated by Doctor Coit, whose influence has been so potent in the development of the movement.

Upon investigation, however, it was found that there was much diversity as to working details and standards of purity, and a conference of the various commissions was held in Atlantic City, June 3, 1907, to discuss the various complicated phases of the work.

This resulted in the organization of a national association which has for its object the affiliation of all medical milk commissions in the United States, the adoption of uniform working methods and standards relating thereto, and the extension of the movement in other cities.

It therefore becomes of interest to study more in detail the organization of these commissions, their exact functions, their working methods and standards, their altruistic motives, and the results thus far accomplished.

THE ORGANIZATION OF MEDICAL MILK COMMISSIONS.

The commissions have in most instances been appointed by local medical societies and practically all of the members were, therefore, physicians. In certain instances, however, commissions have been organized by private clubs or medical societies in cooperation with local business associations and the membership has included men noted for their business acumen and philanthropy.

The opinion now prevails that the medical milk commission should be strictly a medical organization with professional objects for the public good and that the majority of its members should be physicians. It therefore seems desirable that such commissions be created by medical societies and that the members be appointe annually. The membership of the different commissions has usually consisted of 5 to 12 persons, including a chairman, secretary, and treasurer. In two instances at least, notably Cincinnati and Cleveland, representative business men were also included in the membership, thus lending encouragement and support to the work.

It has been suggested that the chief sanitary officer of the locality should be a member ex-officio of each commission and that this body might also act as an advisory to the municipal bureau of milk inspections. Such an arrangement would appear to be of mutual advantage, as the commission might thus lend its influence for the improvement of the general milk supply and in turn be provided with laboratory facilities and relieved of undue expense.

The members receive no pay for their services, but the experts employed by the commissions in making the necessary veterinary inspections, chemical analyses, and bacteriological examinations generally receive fees. In some instances these services have been performed by health department officials or gratuitously by private persons, and the medical inspections are made by members of the commission without compensation.

FUNCTIONS OF THE COMMISSIONS.

The nature of the organization precludes the possibility of supervising the production of more than a limited supply of milk, and this should be of such quality that physicians could unhesitatingly prescribe it for clinical purposes. Different grades of milk, and even cream, have been indorsed in some instances, but the original purpose of the commission would be better subserved by certifying to only one grade of milk, and that as pure as can be produced with our present methods. Such milk should be available also for use in hospitals and milk dispensaries, and it is clearly within the province of commissions to foster its use in such institutions. Special arrangements have therefore been suggested whereby such supply can be certified for delivery in bulk, provided the containers are sealed.

In certain instances, it has seemed wise to assume temporarily the responsibility of indorsing "inspected milk" (with a bacterial count not to exceed 100,000), but this function should properly be performed by the municipal authority who is responsible to the public for the sanitary condition of the general milk supply.

The responsibility of the medical milk commission should be limited to the production of as nearly perfect milk as possible and its reports should be made to the professional body which it represents, only simple statements being supplied to the dairies directly interested.

WORKING METHODS AND STANDARDS.

While the aims and general requirements of the different commissions are similar, there has been considerable diversity in respect to details, most of them, however, of a nonessential character.

The original plan outlined the fundamental requirements, and it remained for each commission to develop working methods and standards suitable to its particular locality.

In any case, the commission agrees to certify to milk conforming to its standards when produced in well-equipped dairies conducted in accordance with prescribed sanitary requirements.

In order that these facts may be determined, a veterinary surgeon, a bacteriologist, and a chemist are selected by the commission.

When a dairyman signifies his willingness to cooperate in the production of pure milk, the veterinary surgeon visits the farm and inspects the buildings, their location, and sanitary condition. He also observes the hygienic methods employed in the production and handling of the milk, and physically examines the cows in the herd.

This examination also includes the application of the tuberculin test. These inspections are subsequently made at frequent intervals and reports are made to the commission, the following being a convenient form in use at Cleveland and other places:

Inspector's report. Dairy of ...... Date ......
Herd: Milking cows ...... Dry cows ...... Hospital cows ......
Cows recently calved ...... Cows added since last report
Not yet tuberculin tested ...... Quarantined ......
Stable: Cleanliness...... Ventilation ...... Temperature ......
Dairy building: Cleanliness ...... Ventilation ...... Temperature ......
Other buildings ......
Utensils ......
Care and cleanliness in milking ......
Food ......
Health of employees ......
Remarks ......

Based upon the foregoing inspection made at the request of The Milk Commission of the city of Cleveland, I beg to report that this dairy conforms to the requirements of said commission and recommend that its milk be submitted to the bacteriologist and chemist for their examinations.

Signed ——— ———, *Veterinarian.*

The chemist and bacteriologist each examine from time to time at the discretion of the commission, samples of the milk taken at random or purchased on the open market.

The former determines the specific gravity, acidity, percentage of fats, sugar, proteids, water and mineral matter present, and the presence or absence of preservatives and chemical adulteration. The latter determines the number, and so far as practicable, the character of bacteria and the presence or absence of pus cells.

The following forms are convenient for rendering the reports of these examinations:

No. ...... Dairy ...... Date ......
Distributer ...... Collected by ......
Sealing ...... Date of milking ......
Hour collected ...... Temperature when collected ...... °F.
General condition: Color ...... Odor ...... Taste ......
Separation of cream ...... Macroscopic sediment ......
Chemical composition: Specific gravity ...... Acidity ...... Total solids ......%.
Fat ......%. Sugar ......%. Proteids ......%. Salts ......%. Ash......%.
Preservatives ...... Coloring matters ...... Adulterants ......
Remarks ......

The examination recorded above made at the request of The Milk Commission of the city of Cleveland, shows a {milk / cream} reaching the chemical standard adopted by the commission.

Signed ——— , *Chemist.*

[The foregoing card is printed on pink paper.]

No. ...... Dairy ...... Date ......
Distributer ...... Collected by ......
Sealing ...... Date of milking ......
Hour collected ...... Cultures made ...... Temperature when examined ......°F.
General condition: Color ...... Odor ...... Taste ......
Separation of cream ...... Macroscopic sediment ......
Bacteriologic examination: Media ......
Temperature ...... Dilution ......
Bacteria per c. c. ...... Average ......
Pathogenic bacteria ......
Microscopic examination, blood, pus, tubercle bacilli, etc. ......

The examination recorded above, made at the request of The Milk Commission of the city of Cleveland, shows a {milk / cream} reaching the bacteriological standard adopted by the commission.

Signed , *Bacteriologist.*

[The foregoing card is printed on light blue paper.]

It is generally believed that the bacteriological examination should be repeated once a week, the chemical examination once a month, and the veterinary inspection once a month—the tuberculin test to be used on every new cow added to the herd and reapplied at least once a year.

Inquiry is also made, usually by a member of the commission regarding the health of employees, and in addition, the dairyman is, in certain instances required to render a regular report regarding the presence or absence of communicable diseases among the dairy personnel. The following form is used at Cleveland, Ohio, for this purpose:

For the information of The Milk Commission, I hereby answer the following questions for the week ending ......, 19 .

I. Are any of the men handling milk at your farm ill with any communicable disease? ......
II. Is there any communicable disease in the families with which they are connected? ......
III. Have any been in contact with any communicable disease and then excluded from the milking place? ....
IV. Shipments of certified milk and cream in past week:
  (a) Quarts of certified milk ...... (b) Pints of certified cream ......
  (c) Bottles of certified milk ...... (d) Bottles of certified cream ......
V. How many unbroken boxes of caps have you? ......

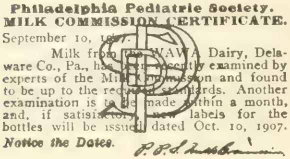
Signed

Upon these reports the commission bases its action in respect to certification and the certificates are renewed once a month.

The dairyman is thus authorized to indicate such indorsement, either by using on his bottle a cap bearing the name of the Medical Milk Commission and the term "certified milk" or a copy of the certificate.

In New York the law forbids the use of the term "certified" on the cap unless accompanied by the name of the society which certifies it. The laws of Kentucky and New Jersey also forbid unwarranted use of the term, and in some other places the certificates bear a copyrighted monogram to prevent their fraudulent use.

The following are examples of these methods of designating such milk:

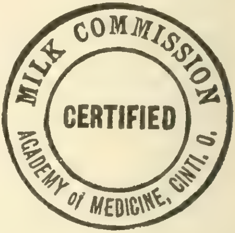


Philadelphia Pediatric Society.
MILK COMMISSION CERTIFICATE.

September 10, 1907.

Milk from the WAWA Dairy, Delaware Co., Pa., has been recently examined by experts of the Milk Commission and found to be up to the required standards. Another examination is to be made within a month, and, if satisfactory, new labels for the bottles will be issued dated Oct. 10, 1907.

Notice the Dates.



When the certificate form is used it is placed between the cap and a parchment covering the neck of the bottle, and in either case the date of milking must appear, a rubber or impression stamp being used for the purpose.

In some instances the bottles are hermetically sealed with paraffin, which is protected by parchment, tin foil or tin covers bearing the term "certified," the name of the dairy and the name of the Medical Milk Commission. The caps are sometimes sold to the dairymen by the commissions and funds are thus provided for defraying the necessary expenses, including inspections, chemical analyses, etc. In other instances funds are provided by the medical society, the dairyman or by means of a bottle tax.

STANDARDS OF PURITY.

The bacterial content of milk has been accepted as the most practical index of the care used in its production and transportation. Bacterial standards for certified milk have therefore been adopted, which limit the number of bacteria and require the absence of pathogenic organisms. The numerical standard is in most cases a maximum limit of 10,000 bacteria per cubic centimeter. The results in different cases no doubt vary somewhat, but with the adoption of established standard methods of technique they should be pretty constant and capable of comparison. The use of heat and preservatives to reduce the number of bacteria are of course forbidden, and to determine the absence of the latter, chemical tests are relied upon.

For the purpose of insuring the constant composition and nutritive value of certified milk, definite chemical standards have been adopted in most cases. Those of the Medical Milk Commission of the Philadelphia Pediatric Society are as follows:

Specific gravity from 1,029 to 1,034; reaction, neutral or faintly acid; proteid from 3 to 4 per cent; sugar from 4 to 5 per cent; fats from 3½ to 4½ per cent; also an additional fat standard of 5 per cent, the permissible limits of variation being from 4½ to 5½ per cent.

A knowledge of the fat content is of much importance, and its determination at regular intervals is required by all commissions. The examination for adulterants and preservatives is of much less importance, as it is altogether unlikely that these substances would be used by dairymen willing to undertake the production of certified milk.

In order to attain these standards great care is necessary in the production and transportation of the milk, and the dairyman is required to observe certain rigid requirements. These are codified and in some instances incorporated in a rigid contract, which is signed by the dairyman and members of the commission.

REGULATIONS OF THE MILK COMMISSION OF THE MEDICAL SOCIETY OF THE COUNTY OF NEW YORK.

The following requirements of the Milk Commission of the Medical Society of the County of New York show great care in preparation, and contain all of the essential rules required by other commissions:

1. *The barnyard.* The barnyard should be free from manure and well drained, so that it may not harbor stagnant water. The manure which collects each day should not be piled close to the barn, but should be taken several hundred feet away. If these rules are observed, not only will the barnyard be free from objectionable smell, which is an injury to the milk, but the number of flies in summer will be considerably diminished.

These flies are an element of danger, for they are fond of both filth and milk, and are liable to get into the milk after having soiled their bodies and legs in recently visited filth, thus carrying it into the milk.

Flies also irritate cows, and by making them nervous reduce the amount of their milk.

2. *The stable.* In the stable the principles of cleanliness must be strictly observed. The room in which the cows are milked should have no storage loft above it; where this is not feasible, the floor of the loft should be tight to prevent the sifting of dust into the stable beneath. The stables should be well ventilated, lighted, and drained, and should have tight floors, preferably of cement, never of dirt. They should be whitewashed inside at least twice a year, unless the walls are painted or of smooth cement finish, which can be washed frequently.

The air should always be fresh and without bad odor. A sufficient number of lanterns should be provided to enable the necessary work to be properly done during the dark hours. The manure should be removed twice daily, except when the cows are outside in the fields the entire time between the morning and afternoon milkings. The manure gutter must be kept in a sanitary condition. All sweeping must be finished before the grooming of the cows begins, so that the air may be free from dust at the time of milking.

There should be an adequate supply of water, warm and cold, and the necessary wash basins, soap, and towels.

3. *Water supply.*—The whole premises used for dairy purposes as well as the barn must have a supply of water absolutely free from any danger of pollution with animal matter and sufficiently abundant for all purposes and easy of access.

4. *The cows.*—No cows will be allowed in the herd furnishing certified milk except those which have successfully passed a tuberculin test. All must be tested at least once a year by a veterinarian approved by the milk commission. Any animal suspected of being in bad health must be promptly removed from the herd and her milk rejected. Do not allow the cows to be excited by hard driving, abuse, loud talking, or any unnecessary disturbance.

Feed.—Do not allow any strongly-flavored food, like garlic, to be eaten by the cows.

When ensilage is fed, it must be given in only one feeding daily, and that after the morning milking, and the full ration shall consist of not more than 20 pounds daily for the average-sized cow. When fed in the fall small amounts must be given and the increase to the full ration must be gradual.

Cornstalks must not be fed until after the corn has blossomed, and the first feedings must be in small amounts and the increase must be gradual. If fed otherwise, ensilage and cornstalks are liable to cause the milk to affect children seriously.

Cleaning.—Groom the entire body of the cow daily. Before each milking wash the udder with a cloth used only for the udders and wipe it with a clean dry towel. Never leave the udder wet and be sure that the water and towel used are clean. The tail should be kept clean by frequent washing. If the hair on the flanks, tail, and udder is clipped close, and the brush on the tail is cut short, it will be much easier to keep the cow clean. The cows must be kept standing after the cleaning until the milking is finished. This may be done by a chain or a rope under the neck.

5. *The milkers.*—The milker must be personally clean. He should neither have nor come in contact with any contagious disease while employed in handling the milk. In case of any illness, in the person or family of any employee in the dairy, such employee must absent himself from the dairy until a physician certifies that it is safe for him to return.

In order that the milk commission may be informed as to the health of the employees at the certified farms, the commission has had postal cards printed, to be supplied to the farms, and to be filled out and returned each week, by the owner, manager, or physician of the farm, certifying that none are handling the milk who are in contact with any contagious disease.

Before milking the hands should be washed in warm water with soap and nail brush and well dried with a clean towel. On no account should the hands be wet during milking.

The milkers should have light-colored, washable suits, including caps, and not less than 2 clean suits weekly. The garments should be kept in a clean place, protected from dust, when not in use.

Iron milking stools are recommended and they should be kept clean.

Milkers should do their work quietly and at the same hour morning and evening. [illegible] increases materially the bacterial contamination of the milk and should be forbidden.

6. *Help other than milkers.*—All persons engaged in the stable and dairy should be reliable and intelligent. Children under 12 should not be allowed in the stable during milking, since in their ignorance they may do harm, and from their liability to contagious diseases they are more apt than older persons to transmit them through the milk.

7. *Small animals.*—Cats and dogs must be excluded from the stables during the time of milking.

8. *The milk.*—All milk from cows sixty days before and ten days after calving must be rejected.

The first few streams from each teat should be discarded, in order to free the milk ducts from the milk that has remained in them for some time and in which the bacteria are sure to have multiplied greatly. If any part of the milk is bloody or stringy or unnatural in appearance, the whole quantity yielded by that animal must be rejected. If any accident occurs in which a pail becomes dirty, or the milk in a pail becomes dirty, do not try to remove the dirt by straining, but put aside the pail and do not use the milk for bottling, and use a clean pail.

Remove the milk of each cow from the stable immediately after it is obtained to a clean room and strain through a sterilized strainer of cheesecloth and absorbent cotton.

The rapid cooling is a matter of great importance. The milk should be cooled to 45° F. within an hour and not allowed to rise above that as long as it is in the hands of producer or dealer. In order to assist in the rapid cooling, the bottles should be cold before the milk is put into them.

Aeration of milk beyond that obtained in milking is unnecessary.

9. *Utensils.*—All utensils should be as simple in construction as possible and so made that they may be thoroughly sterilized before each using.

Coolers, if used, should be sterilized in a closed sterilizer, unless a very high temperature can be obtained by the steam sent through them.

Bottling machines should be made entirely of metal with no rubber about them, and should be sterilized in the closed sterilizer before each milking, or bottling.

If cans are used, all should have smoothly soldered joints, with no places to collect the dirt.

Pails should have openings not exceeding 8 inches in diameter, and may be either straight pails, or the usual shape with the top protected by a hood.

Bottles should be of the kind known as "common sense," and capped with a sterilized paraffined paper disk, and the caps authorized by the commission.

All dairy utensils, including the bottles, must be thoroughly cleansed and sterilized. This can be done by first thoroughly rinsing in warm water, then washing with a brush and soap or other alkaline cleansing material and hot water and thoroughly rinsing. After this cleansing they should be sterilized by boiling, or in a closed sterilizer with steam, and then kept inverted in a place free from dust.

10. *The dairy.*—The room or rooms where the utensils are washed and sterilized and milk bottled should be at a distance from the house, so as to lessen the danger of transmitting through the milk any disease which may occur in the house.

The bottling room, where the milk is exposed, should be so situated that the doors may be entirely closed during the boiling and not opened to admit the mill, nor to take out the filled bottles.

The empty cases should not be allowed to enter the bottling room nor should the washing of any utensils be allowed in the room.

The workers in the dairy should wear white washable suits, including cap, when handling the milk.

Bottles must be capped as soon as possible, after filling, with the sterilized disks.

These regulations in effect provide that none but healthy cows shall be used in the production of "certified" milk, that extraneous contamination of their product shall be reduced to a minimum, that it shall be cooled to 45° F. to prevent bacterial growth, and that it shall reach the consumer before noticeable biological or chemical changes have occurred therein. For their observance the greatest care and intelligence is required, and it is necessary that the dairy be of modern sanitary construction.

This does not imply, however, that elaborate and expensive apparatus is absolutely essential. The surgeon may of necessity be forced to convert the kitchen into a surgical ampitheater, but his skill and attention to detail will insure an aseptic wound. The same in fact is also true of the dairyman, whose conscientious and well-directed efforts will yield, even with limited facilities, a product which may be impossible of attainment in the elaborately equipped dairy of the "agriculturist."

Much also depends upon the zeal of the professional body under whose patronage the dairy operates—a fact clearly evident to one visiting these model establishments.

The sanitary excellence of "certified" milk and the standards it represents may therefore be expected to improve in proportion to the increasing appreciation of the medical profession and the educational attainment of those engaged in dairying and dairy hygiene.

THE RESULTS ACCOMPLISHED.

Since the beginning of the movement seventeen years ago, a limited supply of pure milk has been rendered available for clinical purposes in a number of cities of the country.

The plan which was originated by a member of the medical profession has been the means of arousing that body itself to the importance of pure milk for the use of infants, invalids, and the public generally. In consequence there has been organized the American Association of Medical Milk Commissions, which has held three annual meetings, and which is organized as for a permanent agency in the improvement of milk supplies.

The methods adopted have had an influence in creating a demand for improved conditions in the production of market milk, and in addition exerted a beneficial effect upon the character of the general supply in those localities where "certified" milk is produced. They have also emphasized anew the dangers of bovine tuberculosis and the necessity of preventing the use of milk from tuberculous cows.

The standards of purity have already served as a basis for the formulation of measures which it is proposed to enact into law, a milk conference in the District of Columbia having recommended that "certified" milk be recognized by law and that it be certified by the health officer of the District.

An act was passed by the general assembly of New Jersey and approved April 21, 1909, providing for the incorporation of medical milk commissions and the certification of milk produced under their supervision.

Finally, the continued interest of the medical profession in the sanitary supervision of milk from the farm to the consumer will result in the adoption of new standards of purity far in advance of those in use at the present time.

INFANTS' MILK DEPOTS.

The milk dispensary, or "goutte de lait," was called into existence in consequence of a recognition that bad milk and bad hygiene are responsible for excessive infant mortality among families of the poor.

Its primary object is to encourage maternal feeding, and when this is impossible, to supply a pure milk to meet the special need of the infant. An additional important function consists in the diffusion of knowledge among mothers regarding the hygienic care of their children in the home, especially with reference to the conditions necessary for success in artificial feeding.

The first institution of this character in the United States appears to have been opened by Dr. Henry Koplik at the Eastern Dispensary, New York, in 1889.[a]

During the same year a similar institution was also founded in St. Gertrude's district, Hamburg.[b]

In 1892 Doctor Variot established a "goutte de lait" in connection with the Belleville Dispensary, Paris.

Since 1892 similar establishments have been opened in many localities in this and other countries, either through private philanthropy or governmental agency.

Although the methods employed in the conduct of infants' milk depots have varied somewhat both in this country and abroad, their objects have been the same. It is recognized that all milk dispensed should be produced and transported under conditions insuring a product of the highest purity, that it should be prepared and modified in the depot under medical supervision, and that strict bacteriological precautions should be taken in every step of the process.

In addition to the care exercised in the depot, the milk is packed in a manner to guard against contamination in the home. Each bottle contains but one feeding, and is so designed that it will not stand on end, and therefore can not be left standing open.

The milk is modified in accordance with standard formulæ in use at the various depots, and, in addition, special modifications are made upon the prescriptions of physicians.

The following are formulæ now in use at the Straus milk depots in New York:

FORMULÆ FOR MODIFIED MILKS.

Formula No. 1 (Dr. Arthur R. Green).

Milk	ounces	96
Cane sugar	do	2.5
Salt	do	0.083
Oat water	do	32

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[a] New York Medical Journal, Jan. 31, 1891, and Feb. 4, 1893.

[b] Von Ohlen, Milk Depots in Germany, Public Health, 1905.

Formula No. 2 (Dr. Rowland G. Freeman).

Milk	ounces	64
Limewater	do	4
Milk sugar	do	6
Filtered water	do	60

Formula No. 3 (Dr. A. Jacobi).

Milk	ounces	64
Barley water	do	61
Cane sugar	do	4
Table salt	grains	30

Formula No. 4 (Dr. Rowland G. Freeman).

Cream (16 per cent)	ounces	16½
Milk	do	21½
Milk sugar	do	6½
Limewater	do	4
Filtered water	do	92

Formula No. 5 (Dr. Arthur R. Green).

Cream (16 per cent)	ounces	4
Milk	do	16
Limewater	do	6
Milk sugar	do	6
Filtered water	do	102

The three former mixtures are placed in 6-ounce bottles, the two latter in 8-ounce bottles and pasteurized by exposure of twenty minutes to 157° F. Whole milk is also pasteurized in 8 and 16 ounce bottles.

Practically all infants' milk depots in the United States are under general medical supervision, and, in addition, many depots are in direct charge of graduate nurses who prepare the milk and give instructions to mothers in the care of infants. In some instances, visiting nurses also enter the homes of the children for the purpose of imparting instruction.

With the view of determining the extent of this movement and its influence on the public health, an inquiry was sent by the Surgeon-General of the Public Health and Marine-Hospital Service to the health officials of all cities in the United States containing a population of over 50,000. Replies were received from 65 of the 76 cities in this class, and acknowledgments are due to the health officers and others through whose courtesy the following tabulated information was obtained. The information thus obtained is of much interest and value.

The following table contains a list of the cities in the United States in which infants' milk depots are located, the dates of their establishment, the number of depots operated in each city during the season of 1907, the period of the year in which they were in operation, and by whom maintained:

Cities in the United States having infants' milk depots.	When established.	Number of depots.	Period of year in operation.	Conducted by—
New York, N. Y. . . .	1893	[a]17	7 are open during the entire year; 10 located in parks and on recreation piers are open in summer.	Maintained by Mr. Nathan Straus and conducted by a medical director.
Yonkers, N. Y.	1894	1	June 1 to Sept. 30.	St. John's Riverside Hospital.
Rochester, N. Y	1897	1	July and August.	Health department.
Pittsburg, Pa.	1898	7	May to November; sometimes to December, depending upon the weather. Isolated cases furnished milk during entire year.	Private philanthropy. Pittsburg and Allegheny Milk and Ice Association. Supervised by medical director and secretary of the association.
Cleveland, Ohio	1899	2	Entire year.	Private philanthropy. Milk Fund Association.
Chicago, Ill	[b]1903	28	22 are open during entire year; 6 during summer season only.	Private philanthropy. The Milk Commission of the Children's Hospital Society.
Philadelphia, Pa	[c]1903	20	9 are open during entire year; 11 during summer months.	Private philanthropy. Philadelphia Modified Milk Society. .
Baltimore, Md.	1903	9	8 are open during entire year; 1 during July and August, 1907.	Private philanthropy. Thomas Wilson Sanatorium for Children until 1906. Since then by The Babies Milk Fund Association. Summer station maintained by Playground Association.
St. Louis, Mo	1904	12-15	May to December	Private philanthropy under direction of St. Louis Pure Milk Commission.
Detroit, Mich.	1905	. . . .	At hospital dispensaries. . . .	Private philanthropy. Detroit Milk Fund Association and under medical supervision.
Columbus, Ohio.	[b]1906	1	During entire year	Private philanthropy. Hartman Stock Farm Dairy.
Cambridge, Mass.	1906	5	All open during summer months.	Public appropriations and private philanthropy. Milk inspector with cooperation of Cambridge School of Nursing and Visiting Nurses Association.
Providence, R. I.	1906	5	Open from June 20 to Sept. 6, 1907.	Private philanthropy. Committee of the Providence Medical Association.
Cincinnati, Ohio	1907	2	Open from July 15 to Sept. 1.	Health department, nurse at each station. Physician in general charge.
Jersey City, N. J	1907	5	Opened July 15; to be maintained during entire year	City board of health. Under medical supervision of superintendent, bureau of contagious diseases
Toledo, Ohio	1907	2	1 open during entire year; 1 from June to October.	Private philanthropy.
Kansas City, Mo. .	1907	9	Aug. 1 to Oct. 1.	Private philanthropy. Kansas City Pure Milk Commission.
Kansas City, Kans . .	1907	1	Aug. 1 to Oct. 1	Private philanthropy. Kansas City Pure Milk Commission.
New Bedford, Mass . .	1907	3	July 10 to Sept. 10	Private philanthropy. Charity organization society of New Bedford.
Boston, Mass	[d]	10	5 open during entire year. In addition, out-patient departments of 5 hospitals distribute milk during summer.	Private philanthropy. Hospital work maintained by a "milk fund."
Newark, N. J . .	1899	1	. . . . .	Executive committee, Society of the Babies' Hospital, Newark, N. J.
Brooklyn, N. Y.	1899	16	Summer months . . .	Brooklyn Children's Aid Society.

[a] Depots are also maintained by the Good Samaritan Hospital, The Diet Kitchen Association, and the New York Milk Committee.

[b] April.

[c] July.

[d] First one "many years ago," others about Jan. 1, 1905.

The above table indicates that there are 22 cities in the United States in which infants' milk depots were in operation during the year 1907.

Since the compilation of this table relative to infants' milk depots, information has been received as to other depots showing an extension of the movement.

The general secretary of the Brooklyn Children's Aid Society has invited attention to the work of that society. He has also reported that in New York, in addition to the depots mentioned in the table, there are seven milk stations under direction of the New York milk committee, and one or more each that are maintained separately by the Good Samaritan Dispensary and the Diet Kitchen Association.

While the first depot was organized in New York in 1889, other cities soon took up the work, and during the past two years the movement has progressed rapidly, no less than 6 cities having organized milk depots during 1907.

It is shown that 159 stations were in operation during the present season in the 22 cities from which data was secured, 55 of which will be maintained during the entire year.

In only 1 of the 20 cities were depots maintained at public expense and supervised by officials of the municipalities; all the others owed their existence to private philanthropy, which has set the example in the solution of this great public-health problem, as it has in so many others of economic and vital importance to the State.

The following table contains data regarding the source and character of the milk used, whether it was modified under professional supervision, whether it was pasteurized and the method followed, and the amounts distributed during 1907:

Cities in which are located infants' milk depots.	Source and character of milk used.	Whether modified and by whom.	Whether pasteurized and temperature used.	Amount of milk distributed during present season.
New York, N. Y.	Milk certified by the Medical Milk Commission.	Modifications are prepared in accordance with formulæ of physicians.	All milk is pasteurized by exposure for 20 minutes to a temperature of 157° F. No milk is sold twenty-four hours after preparation.	From Jan. 1 to Sept. 15, 1907, the Straus depots alone dispensed 2,917,336 bottles and 1,222,045 glasses.
Yonkers, N. Y.	Plain milk	Modified under supervision of physician.	Pasteurized by exposure for 20 minutes to temperature of 170° F.	60 quarts daily.
Rochester, N. Y.	Central station in charge of trained nurse located at the farm. Cows are tuberculin tested.	Modified under supervision of a physician.	No	6,000 to 7,000 quarts delivered in nursing bottles ready for use.

Cities in which are located infants' milk depots.	Source and character of milk used.	Whether modified and by whom.	Whether pasteurized and temperature used.	Amount of milk distributed during present season.
Pittsburg, Pa......	Certified milk from Locust Grove Farm	A portion is modified according to physician's prescription at the Walker Gordon laboratory. Work supervised by regular physician.	Modified milk for youngest children pasteurized during period of greatest heat and humidity. Temperature of 157° F. for 20 minutes.	93,417 feedings of modified milk. 27,355 quarts whole milk. Double these amounts dispensed during a year.
Cleveland, Ohio...	Dairy scoring 85..	No..................	No..................	2,700 quarts.
Chicago, Ill	Inspected dairy .	4 standard modifications are used and special modification on physician's prescription.	Yes. Temperature of 165° F. for 20 minutes.	364,126 bottles from Jan. 1 to Sept. 30, 1907.
Philadelphia, Pa ..	Inspected dairy ..	4 standards of modification are used.	All milk is pasteurized by exposure for 20 minutes to a temperature of 180° F.	823,014 bottles from Jan. 1 to Sept. 30, 1907.
Baltimore, Md ...	Burnside Farm...	Modified and bottled by the Walker Gordon laboratory under direction of Dr. J. H. M. Knox, jr.	Pasteurized when air temperature is above 80° F. by exposure for half an hour to temperature of 150° F.	Average for the year is about 1,050 bottles a day.
St. Louis, Mo.....	Certified milk produced under patronage of Pure Milk Commission.	Large proportion modified under direction of chemist, who is under supervision of physician.	Pasteurized by exposure for 20 minutes to temperature of 167° F.	450,000 bottles (2, 4, 5, 7, and 8 ounces).
Brooklyn, N. Y....	....	Yes..................	Yes..................	360,000 bottles.
Detroit, Mich.....	Walker Gordon laboratory	Modified under direction of physician in charge of clinics.	Not pasteurized, except on physician's prescription.	From July to November, 1905, 20,835 bottles of modified milk and 1,367 quarts whole milk.
Columbus, Ohio ..	Hartman Stock Farm Dairy, tuberculin tested herd.	To be modified, beginning in spring of 1908	No...............	Average of 300 babies supplied during first 9 months of 1907.
Cambridge, Mass..	Certified milk....	Yes	No..................	3,387 quarts of milk and 244¼ quarts of cream from Jan. 1 to Sept. 27, 1907.
Providence, R. I. .	Berry Farm	Yes under medical supervision.	No..................	7,413 quarts.
Cincinnati Ohio...	Milk certified by Medical Milk Commission.	No..................	No..	About 4,000 pints distributed among 305 families.
Jersey City, N. J .	Pasteurized milk .	No...	Pasteurized by exposure for 30 minutes to a temperature of 170° F.	About 40,000 bottles from July 20, to Sept. 30, 1907.
Toledo, Ohio	. .....	No	No	About 4,000 quarts.
Kansas City, Mo ..	Hillcrest Farm under best sanitary conditions.	Modified under medical supervision.	Pasteurized by exposure for 20 minutes to temperature of 165° F.	1,800 quarts a month
Kansas City, Kans.	Modified .	4 standard formulæ used, also on special prescription of physicians.	A portion pasteurized by exposure for 10 minutes to temperature of 163° F.	800 quarts of pasteurized milk daily, 40 quarts of modified milk in 5-ounce bottles daily.
New Bedford, Mass	Milk, the average bacterial count of which is 12,000	Modified for young infants by nurses upon prescription of physician.	No..................	Over 7,000 quarts.

Cities in which are located infants' milk depots.	Source and character of milk used.	Whether modified and by whom.	Whether pasteurized and temperature used.	Amount of milk distributed during present season.
Boston, Mass. ..	"Milk fund" supply from Walker Gordon laboratory; the rest from selected country dairies.	Modified "milk fund" modifications made upon prescriptions adapted for each case. The rest is modified according to 3 selected formulæ.	Much of "milk fund" supply is not heated. All milk used by other organizations is pasteurized at temperature of 155° F. for 20 minutes.	"Milk fund" supplied 300 babies in summer of 1906. Other organizations distribute about 1,200 bottles daily.
Newark, N. J.	High grade ..	Yes..................	Pasteurized by exposure to temperature of 155° F.	258,000 bottles to over 500 infants.

In 6 of the above-mentioned cities the supply of milk used in infants' milk depots was certified by Medical Milk Commissions, and in practically all of the others it came from sources of undoubted purity.

In 17 of the 22 cities heard from the milk was modified under medical supervision to meet the special needs of infants.

Pasteurization was practiced in 13 cities for a portion or all of the milk distributed, the temperature used varying from 150° F. to 180° F., and the length of exposure being from ten minutes to thirty minutes. In the other cities pasteurization was not practiced, although in Detroit it was done in special cases upon the prescriptions of physicians.

In Rochester the central milk depot, which was in charge of a trained nurse, was located at the farm, and the herd was tuberculin tested—conditions which should insure the production of pure raw milk.

The extent of the movement may in a measure be estimated by the number of depots in operation and the quantities of milk distributed. The latter, if expressed in the number of bottles delivered, would mount well into the millions.

It is the consensus of opinion of those interested in this work that the results have been exceedingly beneficial, although it is not possible, except in one or two instances, to demonstrate by accurate statistics the reduction in infant mortality. This lack of demonstration is largely due to the fact that the milk was used by a very limited number of the infant population, and to the impossibility of excluding other factors such as environment, climatic conditions, etc., responsible for increased morbidity and mortality. Nor is it necessary to express in numerical terms the value of such institutions. Their educational value alone is sufficient to justify their existence—an influence that has extended both to the consumer and to the subscriber to the milk fund. Mothers have been taught the importance of nursing their infants, and when this was clearly impossible the method and requirements necessary for the successful use of artificial foods.

Through the cooperation of municipal authorities, the medical profession, trained nurses' associations, and others, instruction has also been carried to the homes of infants, with lasting benefit.

There is great necessity for a wider extension of this movement, in order that its benefits may be felt in every congested center of population in the United States. In many of these areas the insanitary conditions surrounding the lives of infants are a menace to the State. Diffusion of knowledge with respect to all that pertains to infant hygiene is therefore demanded.

Mothers should be encouraged in every possible way to nurse their infants, regardless of financial or social status.

When breast feeding is clearly impossible, a pure supply of cow's milk, modified to meet the special needs of the infant, should be rendered available for both rich and poor. At the same time mothers should be instructed regarding the special requirements necessary to successful artificial feeding, including the care and administration of milk in the home.

Private philanthropy has led the way. The public, through its official representatives, should assume its share of responsibility, both because of economic and sanitary considerations, and provide infants' milk depots for improving the physical well-being of the children who are destined to become the active producing members of the community of the future.

21. PASTEURIZATION.

PASTEURIZATION.

By Milton J. Rosenau,

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Pasteurization as applied to milk consists in heating it for a short period of time at a temperature below the boiling point, followed by rapid chilling. As we now understand it, the object is not so much to preserve the milk as it is to destroy the harmful bacteria and their products.

Pasteur in 1860–1864 studied the "diseases" of wine, and found that it was sufficient to heat wine for a few moments at a temperature of from 50° to 60° C. in order to prevent souring and abnormal fermentation. In 1868 the successful experiment was made of sending a cargo of heated wine around the world upon the frigate *La Sybile*.

Following the Franco-Prussian war, Pasteur studied the "diseases" of beer, and found that beer could be preserved by being subjected to a temperature of from 50° to 55° C. The application of this process gave rise to the new term "Pasteurization," which soon became current in technical language.

It was not until 1886 that the distinguished chemist Soxhlet advised the heating of milk for infant feeding and described an apparatus for carrying out the process in the home. To Soxhlet will ever remain the merit of having systematized and popularized the heating of milk for the special use of infants.

In addition to heating the milk, Soxhlet divided the day's quantity conveniently into nursing bottles, which he had caused to be so shaped and arranged as to be readily cleansed and sterilized, upon the importance of all of which he properly laid stress.

Soxhlet made the mistake of regarding milk, heated for a brief period at about the temperature of boiling water, as sterilized. He also placed undue stress upon a special stopper that hermetically and automatically sealed the flasks in cooling.

In America, the Archives of Pediatrics contains no reference to the sterilization of milk until 1888. In 1889 Jacobi,[a] who had long practiced and taught the wisdom of boiling milk for infant feeding, makes reference to the use of Soxhlet's apparatus.

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[a] Arch. Pediat., N. Y., 1889, 1517.

It was soon pointed out by bacteriologists that Soxhlet's process was not sufficient to sterilize the milk, and that the remaining organisms grew and, according to Flügge, were capable of producing harmful results.

Further, it was found that the heating of milk for prolonged periods or at high temperatures was neither necessary nor desirable; and recourse was then had to the pasteurization process. As will be shown later, the confusion between "sterilized" and "pasteurized" milk has been largely instrumental in throwing discredit upon the latter process.

Harm has also been done by the misleading use of the term "pasteurized milk," which has popularly been construed to mean a superior quality of milk, in the same sense that antiseptic surgery is a great advance upon the old time methods. "Pasteurized milk" really means heated milk, and is not necessarily synonymous with "clean milk," "good milk," or "pure milk." The particular object of the heating is to destroy the harmful bacteria. In order to correct this misconception concerning "pasteurized milk," it would be better to discontinue the use of the term and use in its place "heated milk," stating the degree of heat and the time of exposure on each bottle, as well as the date on which the milk was heated.

Pasteurization does not mean simply the heating of milk; the subsequent rapid cooling is a very important part of the process.

If heated milk is cooled slowly it remains at a temperature between 20° and 37° C. for a long time. This is the best temperature for the development of bacteria and their toxic products, and it requires only a few hours under such conditions to produce an enormous growth.

Pasteurized milk must be handled at least as carefully as raw milk, if not more so. Pathogenic bacteria grow more readily in heated than in raw milk. The "germicidal" properties of the milk are destroyed by high heating, and finally the surviving bacteria do not have so hard a struggle for existence in the heated milk. It must not be forgotten that pasteurization kills only the major portion of the nonspore-bearing bacteria, and that a large number of micro organisms remain and, if permitted to grow and multiply, they may occasionally produce undesirable qualities or perhaps poisonous properties in the milk.

It must be quite evident to anyone who gives the matter thoughtful attention that the heating of milk, like the use of antiseptics, is an expedient rather than an ideal procedure. "Antisepsis" was a great improvement in surgery, but "asepsis," or the absence of germs, is the ideal. In the same sense, heating improves bacteria-laden and dirty milk, but clean milk is the end we must seek. "Pure milk is better than purified milk."

Milk presents the strange contradiction of being the most wholesome single foodstuff, and sometimes one of the most poisonous of all foods. A single feeding of a few gills of milk containing pathogenic bacteria or the toxic products of bacterial activity frequently results in sickness and death. Milk sometimes contains such violent poisons as to cause death in a few hours. Ordinarily, milk contains very many bacteria; in fact, milk containing less than 10,000 bacteria per cubic centimeter is considered of excellent quality, and milk containing 100,000 bacteria per cubic centimeter is generally considered good.

Of all foodstuffs, milk is the most difficult to preserve pure and handle with success. It requires not only intelligence, but a high degree of technical training, as well as incessant vigilance, to produce a clean and safe milk. Many believe that this end may be accomplished by official supervision and a good system of inspection. However, we can scarcely conceive of any system of surveillance of the milk supply that will prevent its occasional contamination. In fact, the highest grade of certified milk has at times been accused of causing outbreaks of disease. Such lapses are infrequent and the danger slight. These facts are stated not as an argument that certified milk should be pasteurized, but simply to show the difficulties of obtaining a safe raw product.

Preventive measures are better than corrective ones. Pasteurization can not atone for filth. Milk should be produced under clean conditions and kept clean and it would not then have to be purified. But we must guard against enemies as long as they exist. We would all like to do away with the necessity for armies and navies, but present conditions demand their maintenance. The same is true of harmful bacteria in milk; so long as the average market milk is apt to contain these insidious foes, the only protection we have is to destroy them with heat.

There can be no more objection to the heating of milk for the use of adults and of children above the age of 3 years than there is to the cooking of meat. Even Flügge,[a] who was one of the first to sound the warning that heated milk may subsequently develop poisonous properties for infants, has no objection to the heating of milk for the use of adults and of children above the age of 3 years.

The question naturally arises, Is the danger from the use of the average raw market milk a serious one? Our investigations[b] in Washington have shown that the general market milk is, for the most part, old, stale, and dirty (in 1907 the milk averaged over 22,000,000 bacteria per cubic centimeter and in 1908 over 11,000,000), and further, that at least 11.3 per cent of the cases of typhoid fever which occurred during the summer of 1906 in Washington were certainly attributable to contaminated milk. In 1907 9.18 per cent and in 1908 about 10 per cent of the typhoid cases were traced to infected milk. Similar conditions have been found in other cities wherever the matter has been investigated. In addition to typhoid fever, the milk frequently conveys the infection of tuberculosis, scarlet fever, diphtheria, diarrhoeal and other diseases. One needs only to refer to other parts of this bulletin to assure himself of the extent to which death and disease are caused by impure milk. That phase of the subject therefore needs no further emphasis here.

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[a] Flügge: Die Aufgaben und Leistungen der Milchsterilisirung gegenüber den Darmkrankheiten der Säuglinge. Zeit. f. Hyg., vol. 17, 1894, p. 272.

[b] Rosenau, Lumsden, and Kastle, Bulls. 35 and 44, Hyg. Lab., U. S. Pub. Health and Mar. Hosp. Serv., Wash., 361 pp.

The average commercial milk of large cities is not a safe food. The principal reasons for this are the ignorance and indifference of those engaged in the dairy business, filthy barns, unclean and unhealthy cows, improper care of containers, insufficient cooling of the milk, long transportation, unnecessary and frequent handling, imperfect cleaning and lack of sterilization of the bottles, and the frequent close association with contagious disease.

The difficulty of obtaining a clean fresh milk supply is soon appreciated when we investigate the subject in any large city. For instance, in Washington the milk supply comes from over 1,000 different dairy farms, situated in the surrounding counties of Virginia and Maryland. Some of the cream comes from distant points in Pennsylvania and New York. Boston gets a large part of its milk supply from distances of 40 to 100 miles. The milk supply of the city of New York is produced at 35,000 farms scattered over 5 different States, passes through 400 creameries, and comes over 12 different lines of transportation. Some of the milk, at certain seasons, reaches New York from Canada, and shipments of cream arrive daily from Ohio. One hundred and fifty wholesale dealers are engaged in the business and the retail stores number 12,000, the daily consumption being 1,500,000 quarts. From this extreme case we will find every grade of complexity down to the small village and the individual farmhouse where fresh milk may be obtained twice daily. In New York only 16,000 quarts of the total of 1,500,000 quarts daily used are "certified" as clean milk.

THE EXTENT OF PASTEURIZATION.

Freeman tells that it was about 1892 that the sterilization of the milk in the tenements of New York was widely adopted. So general has this become that the inspectors of the Rockefeller Institute for Medical Research, when recently seeking statistics concerning the effect of different sorts of food on the health of babies in the tenements, were able to find scarcely any infants that were fed on raw milk.

It is now estimated that about 25 per cent of the total daily milk supplied to the city of New York is pasteurized.

About 123,250 of the total of 368,489 quarts of milk which come to Boston daily are subjected to commercial pasteurization.

Pasteurization in bulk is practiced on a large scale in the creameries of Europe, particularly in Denmark and Germany. In Berlin and Copenhagen, especially, commercially pasteurized milk is in general use. In Denmark, in fact, paragraph 6 of the law of March 26, 1898, relating to measures for combating tuberculosis in cattle and hogs, requires that all skimmed and bottled milk from Danish dairies to be used for feeding animals must first be heated to 85° C. This law, which went into effect June 1, 1899, was revised in 1903, and again on February 5, 1904, by requiring the products to be heated to 80° C. and adding to the products requiring pasteurization, cream used for the manufacture of export butter. Paragraph 7 of the same law requires that only such milk and buttermilk may be brought into Denmark as has been heated to at least 80° C. The Minister of Agriculture is, however, permitted to make certain exceptions.[a]

In France the heating of milk is practiced by the wholesale dealers who supply Paris. A portion of the milk sold in certain of the larger cities of France and of the milk distributed from the milk depots ("gouttes de lait") is also first heated. Much of the cream destined for Paris is pasteurized.

LAWS AND REGULATIONS CONCERNING PASTEURIZATION.

Recently the State of Massachusetts and the cities of New York and Chicago have adopted measures relating to the pasteurization of milk and milk products.

Massachusetts.—There are no regulations of the health department of the city of Boston covering the pasteurizing of milk and milk products, but in the year 1908 a State law was adopted. Jordan considers the measure an inoperative one because of the high temperature specified therein. The Massachusetts State act follows:

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[a] Adolf Reitz, Milchhygiene u. Tuberkulosebekampfung in Danemark u. Schweden, Zugleich ein Beitrag zur Technik Pasteurisierapparate. Zeit. f. Fleisch- u. Milchhygiene, 1905 6, 16, p. 143.

ACTS OF 1908, CHAPTER 570.

HEATED MILK.

SECTION 1. Whoever, himself or by his servant or agent, or as the servant or agent of any person, firm, or corporation, sells, exchanges, or delivers, or has in his custody or possession with intent to sell, exchange, or deliver any milk which has been subjected to artificial heat greater than one hundred and sixty-seven degrees Fahrenheit, not having the words "heated milk" distinctly marked upon a light ground in plain black uncondensed gothic letters at least one inch in length, in a conspicuous place upon every vessel, can, or package from or in which such milk is, or is intended to be, sold, exchanged, or delivered shall for a first offense be punished by a fine of not less than fifty nor more than two hundred dollars, for a second offense by a fine of not less than one hundred nor more than three hundred dollars, and for a subsequent offense by a fine of fifty dollars and by imprisonment for not less than sixty nor more than ninety days. If such vessel, can, or package is of the capacity of not more than two quarts, said words may be placed upon a detachable label or tag attached thereto, and said letters may be less than one inch in length, but not smaller than brevier gothic capital letters.

SEC. 2. Nothing in this act shall be construed as applying to condensed milk, or to milk which has been concentrated to one-half its volume or less.

Chicago. The following rules, regulating the pasteurization of milk and milk products, have been adopted by Dr. W. A. Evans, commissioner of health of the city of Chicago:

RULES REGULATING THE PASTEURIZING OF MILK AND MILK PRODUCTS.

The following rules shall regulate the pasteurizing of milk and milk products offered for sale, exposed for sale, or kept with the intention of selling within the city of Chicago, after January 1, A. D. 1909:

RULE 1. *Milk and skimmed milk.*—Milk and skimmed milk shall not contain more than 100,000 bacteria per cubic centimeter from May 1 to September 30, and not over 50,000 bacteria per cubic centimeter between October 1 and April 30.

RULE 2. *Cream and ice cream.*—Cream and ice cream shall not contain more than 200,000 bacteria per cubic centimeter from May 1 to September 30, and not over 100,000 bacteria per cubic centimeter between October 1 and April 30.

RULE 3. *Milk, skimmed milk, buttermilk, cream, and ice cream.* An original package of pasteurized milk, skimmed milk, buttermilk, cream, or ice cream, exposed to the temperature of the room for forty-eight hours and stoppered with a sterile cotton plug, shall not show evidences of putrefaction, after being so exposed.

RULE 4. *Skimmed milk and ice cream.* Skimmed milk and ice cream shall give a negative test when treated in the following manner:

To 5 c.c. of the pasteurized product add two drops of a 2 per cent solution of paraphenylenediamin, and one drop of a 2 per cent solution of hydrogen peroxide, and agitate. Not more than a tinge of blue shall be obtained by this test within thirty seconds after mixing.

RULE 5. *Butter.* Butter shall respond to the following test:

Twenty-five grams of pasteurized butter placed in a small beaker and heated by being placed in water at 60° C., the clear butter fat then poured off and the remaining liquid then diluted with an equal volume of water. The mixture thus obtained is now treated with two drops of a 2 per cent solution of paraphenylenediamin and one drop of a solution of 2 per cent hydrogen peroxide. When thus treated not more than a perceptible blue color shall be obtained within thirty seconds after mixing.

RULE 6. *Pasteurizing temperatures.*—All pasteurized milk, cream, skimmed milk, milk products, and milk and cream used in the production of milk products shall be pasteurized in accordance with the following regulations:

(A) Continuous pasteurization: In all continuous pasteurization the milk and cream shall be heated to a temperature which shall be determined and fixed by the department of health for each machine at a point corresponding to the temperature required to kill 99 per cent of the bacteria and all pathogenic bacteria contained in the raw product. For this determination ordinary raw milk containing in the neighborhood of 3,000,000 bacteria shall be used and the pasteurized product shall be collected as it flows from the cooling apparatus.

All continuous pasteurizers shall be equipped with a feeding pipe which is so constructed that the pasteurizer can not be fed in excess of its normal working capacity; that is, in excess of the working capacity of the machine at which 99 per cent of the bacteria are killed when the required amount of heat is applied.

All continuous pasteurizers operated outside of the city limits, for the production of pasteurized milk and milk products to be sold in the city of Chicago, shall be equipped with an apparatus regulating automatically the supply of steam and heat, so as to correspond with and produce the required temperature of the outflow of the pasteurized product. These automatic thermoregulators shall be accurate, and must be approved by the commissioner of health before being installed.

A recording apparatus shall be installed upon all continuous pasteurizers operated within the city limits so as to record during operation the temperature of the pasteurized product as it flows from the heater. The thermometer of this recording apparatus must be accurate and kept emerged in the milk in such a way that it is not exposed to escaping steam or other heat, except the heated milk.

The records made by this recording thermometer must be accurate and made in a chamber which is kept under lock and key in the control of the department of health.

The automatic thermo regulating and recording apparatus may be combined into one instrument, and it is recommended that all pasteurizers be equipped with both appliances or the combination apparatus.

(B) Held pasteurization: Whenever milk is held during pasteurization in such a manner that the process of pasteurizing is not a continuous one, namely, a continuous flow of milk through the heating or heat-retaining chamber, the process shall be designated as "Held pasteurization." Such methods of pasteurization and pasteurization appliances or systems installed and used shall be examined and approved by the commissioner of health, or his duly appointed representatives, when all of the following requirements are fulfilled:

1. When the pasteurized product shows that over 99 per cent of the bacteria and all pathogenic bacteria contained in the raw product have been destroyed.

2. When the mechanism of the pasteurizer or pasteurizing system is such that the three important elements, namely, the temperature, time of exposure, and the quantity of milk exposed at one time, can be readily kept under control and observation by the department of health.

3. When the following conditions are complied with

A uniform heating of 140° F. maintained for twenty minutes; 150° F. maintained for fifteen minutes; 155° F. maintained for five minutes; 160° F. maintained for one and one-half minutes; 165° F. maintained for one minute.

The time shall be calculated from the period that the entire quantity reaches the required temperature.

RULE 7. *Cooling temperatures.*—The pasteurized product shall be cooled at once to a temperature of 45° F. or less. This cooling shall be so conducted that the pasteurized product is not exposed to the air or other contamination. This cooling apparatus shall be so constructed that it can be readily cleaned and sterilized.

New York.—In the amendments of the sanitary code of the department of health of the city of New York additional regulations for the sale and care of milk were adopted April 22, 1908. Among the additions the following rules apply to pasteurization:

PASTEURIZED.

1. Pasteurization of milk must be carried out under a permit therefor issued by the board of health, in addition to the usual permit for milk required by section 56 of the sanitary code.

2. The milk after pasteurization must be at once cooled and placed in sterilized containers and the containers sealed.

3. All pasteurized milk must be delivered to the consumer in sealed containers which are plainly labeled "Pasteurized." The labels must also bear the date and hour when the pasteurization of the milk was completed, the degree of the heat employed, the length of time exposed to the heat, and the number of the pasteurization permit issued by the board of health.

4. Pasteurized milk must be delivered to the consumer within twenty-four hours of the pasteurization.

5. No milk shall be pasteurized a second time.

CHANGES IN THE MILK PRODUCED BY HEATING.

The changes produced in milk by heating depend upon the degree of heat and the length of exposure. The exposure of milk to a temperature of 60° C. for a short time does not appreciably affect its chemical and physical properties. The boiling of milk, however, produces pronounced changes. These changes consists mainly of the following:

Decomposition of the proteins, and other complex nitrogenous derivatives; diminution of the organic phosphorus; increase of inorganic phosphorus; precipitation of the calcium and magnesium salts and the greater part of the phosphates; expulsion of the greater part of the carbon dioxide; caramelization or burning of a certain portion of the milk sugar (lactose), causing the brownish color; partial disarrangement of the normal emulsion and coalescence of some of the fat globules; coagulation of the serum albumin, which begins at 75° C.

The casein is rendered less easy of coagulation by rennin and is more slowly and imperfectly acted upon by pepsin and pancreatin. Boiling gives the milk a "cooked" taste. The cream does not rise well, if at all.

When the milk reaches about 60° C., a scum forms on the surface which consists of—

	Per cent.
Fatty matter	15.12
Casein and albuminoid	50.86
Ash	3.72

Milk heated in closed vessels does not form a pellicle even when the temperature reaches 100° or 110° C. Milk heated in the open air, after cooling forms a pellicle on the surface which renews itself if it is removed. It seems that this pellicle is due mainly to the drying of the upper layer of the liquid. The cream probably does not rise well in heated milk, owing to the increase in the viscosity of the liquid in which it is emulsified.

Heat kills the ferments in milk,[a] which according to some authors play a useful rôle in digestion and metabolism. We have no direct knowledge of the utility of these milk ferments. For the child to digest and assimilate cow's milk to advantage the complex albuminous substances must first be broken down by the processes of digestion into simpler products and again synthetized. In other words, cow proteins must be converted into human proteins. In this process ferments play an essential rôle. We know that the digestive tube contains ferments that dissolve and break up the complex proteins into simpler substances, but concerning the rearrangement of the molecular structure into the form best suited for assimilation we have little definite knowledge. While ferments play an active part in both the breaking down and the building up processes, it remains for future investigation to determine which particular ferments are helpful in the latter process. It has been abundantly shown by laboratory work that the ferments in milk, or most of them, at least, can withstand a temperature ranging from 60° to 65° C. for some time without material injury. Between 65° and 70° most of these are weakened in their activity, and between 70° to 80° all of them are destroyed, even after relatively short exposure. (Kastle.)

Raw milk shows the peroxidase reaction, whereas milk which has been heated for one hour at 70° C., or for shorter intervals at higher temperatures, does not exhibit this reaction. In this connection Kastle and Porch have observed that on heating milk to 60° C. for 20 minutes, the peroxidase reaction of many specimens of milk is not only not diminished but if anything somewhat intensified.

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[a] Hippius (Deut. med. Woch., vol. 27, 1901, p. 481, 502) states that the oxidizing ferments are able to withstand temperatures between 60° and 65° C. for a long time, but are destroyed after a short exposure to 76° C. The lipase, or fat splitting ferment, withstands one hour's heating at 60° C., or 62° for a short time; is weakened at 63°, and destroyed at 64° C. The proteolytic ferment withstands one hour's heating at 60° or half an hour at 65° C. The amylase withstands one hour at 60° and is only destroyed at 75° C. (See also Kastle and Roberts's article, No. 10, p. 313, this bulletin.)

The heating of the milk produces a decomposition of the albuminoid matter, manifesting itself by the production of a little hydrogen sulphid. This gas may also be produced by the action of micro-organisms.

It is claimed that the heating of milk renders a part of the phosphates insoluble, and that this change favors rachitis in children artificially fed with it. On the other hand it appears to be the general opinion of physicians that rachitis is the result of defective alimentation, due to causes other than the changes in heated milk.

The heating of milk for half an hour at a temperature of 150° F. (65° C.) or over, has the effect of entirely preventing the rising of the cream or of delaying it very materially. In normal milk the larger proportion of the fat droplets agglutinate into tiny globules or masses. At a temperature of 65° C. or above, these clusters are broken down and the globules are more homogeneously distributed throughout the fluid.

The cooked or scalded taste appears at about 70° C., and becomes more pronounced the higher the temperature. This is due perhaps to changes in the nitrogenous products in the milk. The loss of certain gases also alters the taste, so that milk heated in closed vessels has a much less pronounced flavor than if heated in open vessels.

Milk sometimes curdles in the process of pasteurization. This is due to the amount of acid and calcium salts which it contains. In order to avoid such accidents, Kastle advises that the only safe rule to follow is to determine the effect of heating on small samples of the milk, which it is proposed to pasteurize.

It has been observed that cooked milk coagulates with rennin more slowly than raw milk. This effect is noted often at temperatures of 80° to 90° C., but it has not been observed in milk heated to 60° for twenty minutes. The curd produced by rennin coagulation in cooked milk is softer, less tough, and more flocculent than that produced by rennin coagulation in raw milk. This is believed to be an advantage favoring the digestibility of heated milk.

TEMPERATURE AND TIME OF HEATING.

The two dominant factors that control the temperature and time at which the milk should be pasteurized are, (1) the thermal death points of pathogenic bacteria, and (2) the ferments in the milk. The first must be surely killed so as to eliminate this danger, and the second should not be affected sufficiently to "devitalize" the milk.[a]

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[a] Reference to the article upon "The germicidal property of milk," Rosenau and McCoy, p. 455, shows that a temperature of 60° for twenty minutes but slightly affects this property of fresh raw milk. In old milk the so-called "germicidal action" disappears spontaneously.

So far as we are able to judge from our present knowledge the best temperature is 60° C. continued twenty minutes. A higher degree of heat for a shorter time is just as effective so far as the destruction of the bacteria is concerned.

It may be confidently stated that the tubercle bacillus and the specific micro-organisms causing typhoid fever, diphtheria, dysentery, cholera, etc., are rendered harmless by heating to 60° C. for twenty minutes. This opinion is based not only upon experimental data which have been obtained in the Hygienic Laboratory and recently published[a] but upon the experience and experiments of many others who have investigated this subject. It is fortunate that the thermal death points of the pathogenic bacteria that most concern us are below those of the ferments in milk, for in this way all infectiousness may be destroyed without "devitalizing" the milk.

So far as the true bacterial toxines are concerned, our knowledge is not so precise. We know that the true bacterial toxines are thermolabile: that is, readily affected by heat. Kitasato[b] showed that 65° C. and above is sufficient to destroy tetanus toxine in five minutes or less. It will sometimes withstand 60° C. for ten minutes, but is destroyed at 60° C. for twenty minutes.

Diphtheria toxine is also rendered almost inert at about 60°, and botulism toxine is almost equally sensitive to heat.

There is a group of bacterial poisons, however, which resists high temperatures. For instance, Marshal and Gallston found that heating the cell substance of *B. coli commune* to 134° C. for fifteen minutes did not appreciably lessen its toxicity. Cooley and Vaughan heated the same substance in a sealed tube to 164° C. without rendering it inert. Vaughan states further in his recent Shattuck lecture that many crude bacterial poisons withstand the boiling temperature. The nature of these poisons is not known. They are obtained by laboratory devices, and similar substances have never been found in market milk.

It must further be observed that the poisonous properties of all these substances have mostly been determined by inoculation experiments and not by feeding. It can not be denied that milk may at times contain heat-resisting poisons, but their existence has been inferred, not demonstrated. Emphasis is placed upon this seemingly inconsequential point, for the reason that one of the principal objections to pasteurization has always been that the heat does not necessarily destroy the poisonous products of bacterial activity. If such stable poisons are present in milk, and heat does not render them inert it is a limitation, not a disadvantage of the process. If heat-resisting poisons are present in milk, the raw product will be quite as toxic as the heated, probably more so, for the heat may check the further production of such substances by its destructive action upon bacteria.

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[a] Hygienic Laboratory Bulletin 42, "The thermal death points of pathogenic micro-organisms in milk," by J. M. Rosenau, Washington, 1908.

[b] Kitasato, S.: Experimentelle Untersuchungen über das Tetanusgift. Zeit. f. Hyg., vol. 10, 1891, p. 267.

At first, "sterilization" at or above the boiling point was attempted. It was soon shown that it was exceedingly difficult to sterilize milk on account of the resistant spores, and further, that a high degree of heat is not necessary. A more precise knowledge of the objects to be attained has gradually resulted in lowering the temperature and shortening the time. Temperatures varying from 95° to 60° C., and periods varying from a moment to two hours, have been variously recommended for the pasteurization of milk. As a rule the controlling factor is the thermal death point of the tubercle bacillus.

The temperature and time determined by various authorities for milk pasteurization follows:

Authorities.	Year.	Temperature (°C.).	Time (minutes).
Freeman[a]	1898	68	20
Freeman[b]	1907	60	40
Forster[c]	1892	70	5-10
Smith, Th.[d]	1899	60	20
Oppenheimer[e]	1899	70	30
Hippius[f]	1905	60	60
Bitter[g]	1890	68-69	30
Hesse[h]	1900	60	15-20
Russell & Hastings[i]	1900	60	20

[a] Freeman, Arch. Pediat., N. Y. (1898), v. 15, p. 514.
[b] Freeman, Jour. A. Med. Assn., Vol. XLIX, Nov. 23, 1907, 21, p. 1740.
[c] Forster, Hyg. Rundschau, Berl. (1892), v. 2 (20), 15. Okt., p. 872.
[d] Smith, Th., J. Exper. Med., N. Y. (1899), v. 4, p. 233.
[e] Oppenheimer, Münch. med. Wchnschr. (1899), v. 46, p. 1462.
[f] Hippius, Jahrb. f. Kinderh. (1905), v. 61, pp. 365–384.
[g] Bitter, Ztschr. f. Hyg., Leipz. (1890), v. 8, p. 255.
[h] Hesse, Ztschr. f. Hyg., Leipz. (1900), v. 34, p. 347.
[i] Russell and Hastings, 17 Ann. Rep., Agric. Exper. St., Univ. Wis. (1900), p. 170.

In view of certain differences of opinion concerning the temperature and time of milk pasteurization, the definition still lacks completeness. Therefore the misconceptions and confusions concerning the use of the term "pasteurized milk" have added to the prejudice against the process. *We should protest against a word which means a generality and again insist upon all pasteurized milk being properly labeled with the degree of heat, the period of time, and also with the date on which it was subjected to the process.*

So far as we may conclude from the evidence at hand, the heating of milk to 60° C. for twenty minutes destroys pathogenic microorganisms without injuriously affecting its composition or quality and without sensibly hurting its food value. We have authority for the statement that milk pasteurized at 60° C. for twenty minutes is "live" milk, rich in zymogens, and that such milk retains entirely the taste of fresh milk and is quite as digestible.

THE BACTERIA AND TOXINS CONCERNED.

Despite the great amount of work done upon this subject, there is a diversity of opinion as to which particular varieties of bacteria and their varied products are responsible for the large group of diseases comprised under the term "gastro-intestinal infections." There can be no doubt that there is a direct relation between the bacteria and their products in milk and the bowel complaints of children. It is also clear that these are not all acute specific diseases due to one cause. The factors are complex. It is not only the bacteria and their poisons in the milk, but also the bacteria always contained in the gastro-intestinal canal, that play an important part. While it is undoubtedly true that milk sows the seed and often actually contains the poison, it is also well known that a deranged digestion, which favors abnormal fermentation and putrefaction of the milk within the body, resulting in the class of affections known as "auto-infections" and "auto-intoxications," here plays a definite rôle. All clinicians agree that the first essential for the successful treatment of the gastro-intestinal diseases of children is to at once discontinue the use of milk. The great prevalence of this class of diseases in the heated months of summer makes it perfectly plain that the depressing influences of heat seriously affect the resistance of the infant. At the same time the heat favors the growth and multiplication of the bacteria in the milk.

Children vary much in their susceptibility to the bacteria and the bacterial products concerned. The same milk may act as a violent poison to one child while another living under the same conditions may escape.

Flügge[a] laid particular distress upon the peptonizing bacteria which for the most part are spore-bearing organisms. The spores survive the heat of pasteurization and have a free field for growth and activity. As a rule the organisms known as the lactic acid group gain the ascendancy in raw milk, and these bacteria have a restraining effect upon the great majority of other species. Flügge found 3 of the 12 peptonizing bacteria isolated by him from heated milk to have poisonous properties. Pure cultures in milk, when injected into laboratory animals, cause severe symptoms, and in one instance when fed to a puppy produced fatal diarrhea.

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[a] Flügge, C.: Die Aufgaben und Leistungen der Milch Sterilisirung gegenüber den Darmkrankheiten der Säulinge. Zeit. f. Hyg., vol. 17, 1894, p. 272.

In a recent article Colwell and Sherman[a] point out that the pasteurization of milk at 60° C. appears to restrain peptonization to about the same extent that it restrains souring. It apparently has no constant effect in rendering the milk either more or less liable than raw milk to the development of offensive odors.

Following Flügge, attention was focused upon the peptonizing action of bacteria. The evidence is contradictory, but for the most part, Flügge's contentions have not been confirmed. Recent investigations show that the cleavage of proteids by bacteria is much more like that caused by the digestive juices than has heretofore been supposed. There is little evidence for the view that poisonous substances can be formed by the direct cleavage of proteids by bacteria; in fact, the two best known of the bacterial poisons (diphtheria and tetanus toxines) can be produced in proteid-free media.

The bacterial toxines.—Much has been written upon "toxines" in milk. However, when we sift the matter down we find that we know practically nothing of the true bacterial toxines concerned.

Toxines are soluable chemical substances of unknown composition that produce poisonous symptoms after a definite period of incubation and are capable of inducing immunity as a result of the production of antibodies.

We are acquainted with very few bacterial toxines. The best examples are tetanus, diphtheria, and the toxine produced by the *Bacillus botulismus*. These toxines are not resistant to heat; they are all rendered practically inert at a temperature of 60° C.

Bacterial toxines are not the result of proteolytic action upon the albumins contained in the media in which they grow. They may be secreted by, or result from, the breaking down of proteids of the bacterial cell. Concerning their mode of production and their chemical nature we have no definite knowledge.

None of the true toxines are poisonous when taken by the mouth except the botulism toxine. For instance, the strongest one known (tetanus) is harmless when taken by the mouth.[b] We have given a guinea pig 24,000 and a mouse 8,000 minimal lethal doses by the mouth without appreciable effect. It is, therefore, plain that the effects of a toxic substance found in milk when injected into laboratory animals is no criterion of its effect when taken by the mouth.

Particular attention is drawn to this fact because much of the experimental work upon the poisonous substances in milk has been done by injecting these substances into the tissues of lower animals. It is now plain that violent poisons, when introduced subcutaneously, may be inert when taken by the mouth. We must also be cautious in interpreting feeding experiments upon lower animals as applied to man, especially when we consider the great differences in susceptibility of the gastro-intestinal tracts of different species. This difference is marked even among infants, for it is known that not all persons taking poisonous milk suffer equally, and some escape altogether.

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[a] Colwell, R. H., and Sherman, H. C.: "Chemical evidence of peptonization in raw and pasteurized milk." Journ. Biolog. Chem., Oct., 1908.

[b] Snake venom is also harmless by the mouth.

The "endotoxines" and bacterial proteids are substances which are more or less firmly retained by the living bacterial cells. The poisonous action of these so-called "toxines" is closely associated with the phenomenon of anaphylaxis.[a] We are not yet sufficiently well acquainted with the composition and mode of action of this important class of poisons to formulate their relation to milk.

The colon group.—The type of this large group of bacteria was first described by Escherich as *Bacillus coli commune.* While the colon bacillus is undoubtedly the cause of certain pyogenic and septicemic conditions, its power to produce harm in milk is uncertain. Normally it is practically always present in the lower intestines of mammalian animals, where it doubtless serves a useful purpose perhaps by keeping harmful varieties in check.[b]

The colon bacillus was at one time regarded as the common cause of various diarrheal infections, but it has now been differentiated into the dysentery bacillus, the typhoid bacillus, and other closely allied species. Even now it is difficult to disassociate its action from that of its closely allied cousin, the *B. lacticus aerogenes.* These two organisms sometimes induce excessive fermentation of lactose and other sugars with the production of irritating acids (especially acetic and lactic) and at the same time liberate an excessive amount of gas, thereby causing diarrhea.

The typhoid bacillus.—It is known that this bacillus often contaminates milk, which thus becomes the vehicle of some of the typhoid fever in large cities. The paratyphoid and paracolon organisms are closely allied and may doubtless be transmitted in like manner. The paratyphoid bacillus is a frequent cause of meat poisoning, but a similar action in milk has not been shown.

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[a] Rosenau, M. J., and Anderson, John F.: "A study of the cause of sudden death following the injection of horse serum." Bull. No. 29 Hyg. Lab., U. S. Pub. Health and Mar. Hosp. Serv., Washington, 1906.

Rosenau, M. J., and Anderson, John F.: "Further studies upon hypersusceptibility and immunity." Bull. No. 36 Hyg. Lab., U. S. Pub. Health and Mar. Hosp. Serv., Washington, 1907.

Also Hyg. Lab. Bulls. Nos. 45 and 50, upon the same subject.

[b] For a full discussion of intestinal bacteria and their products, see Herter's admirable book on the Common Bacterial Infections of the Digestive Tract and the Intoxications Arising from Them. 360 pages. New York, 1907.

The dysentery bacillus.—Shiga, who discovered this organism, now recognizes 5 types, based on fermentative changes. This organism, like the typhoid bacillus, is "hemiparasitic" in the sense used by Herter; that is, it produces disease only when the bacilli have been introduced in considerable numbers or have had an opportunity to multiply owing to the feeble powers of resistance on the part of the infected individual.

It seems that the nearer the various varieties of dysentery and typhoid bacilli approach the colon bacillus the less virulent they become.

The dysentery bacillus has only been known since 1898 and its relation to milk is not well worked out, but there can be little doubt that milk may by a means of spreading infection by this organism.

The Bacillus proteus or proteus vulgaris.—This is a common organism found frequently in normal feces in moderate numbers and commonly contaminates milk. This bacillus produces the tryptic ferment that peptonizes casein and it also attacks carbohydrates. That this organism may induce acute diseases of the gastrointestinal tract appears to be well established.

The tubercle bacillus.—Tubercle bacilli have frequently been found in milk, and their relation to disease is discussed elsewhere.

Koch's cholera bacillus.—The specific cause of cholera requires an alkaline medium in which to grow. As milk is usually acid or soon becomes acid, the cholera vibrio has little chance of survival, though small outbreaks of cholera have been traced to milk.

The Micrococcus melitensis.—This organism causes Malta fever and is found in goats' milk. It is fully discussed elsewhere in this bulletin.

The Bacillus diphtheria.—The diphtheria bacillus finds favorable conditions for growth and multiplication in milk. A number of outbreaks of diphtheria have been traced to milk so contaminated.

Streptococci and Staphylococci.—These form an exceedingly important group of organisms on account of their frequent and exceptional virulence. They are almost constantly found in milk, frequently in great numbers. Herter says that the human intestinal tract under normal conditions is probably most ot the time free from pathogenic varieties of this group of cocci.

In healthy adults these pathogenic bacteria introduced with milk are ordinarily quickly destroyed in the upper portion of the tract. During infancy the digestive tract is very much less resistant to streptococcic infection. An invasion of the mucous membrane by streptococci is of frequent occurrence and may be associated with disturbances of almost any grade of severity.

It has been shown by Bucher in this country, and by Escherich of Germany, that some of the severest forms of infantile ileo-colitis are associated with streptococcic infections and are probably dependent upon them.

The *Streptococcus lactis*, first described by Kruse, has been proved by Heinemann in this country to be one of the common causes of lactic acid fermentation in milk. It appears to be indistinguishable from the pathogenic forms and is always present in market milk.

The anaerobic spore-bearing micro-organisms.—Flügge first pointed out the importance of the anaerobic spore-bearing organisms in milk and their relation to infantile diarrheas. He especially singled out the *B. butyricus* (Botkin). It now appears that Botkin's bacillus represents two distinct micro-organisms. Herter considers that the *B. putrificus* and the *B. aerogenes capsulatus*, which grow in milk, play an important rôle in intestinal putrefaction. The *B. aerogenes capsulatus*, for instance, produces poisons belonging to the hemolytic and proteolytic class. According to Kamen,[a] it also forms soluble poisons obtainable by filtration. Kamen likens this poison to "sepsin," in that it acts as a respiratory poison and induces vomiting, diarrhea, tenesmus, and death. This poison is not destroyed by heating to 60° for fifteen minutes.

In addition to the bacteria and the bacterial products above considered, the products of fermentation and putrefaction in milk have long been regarded as poisonous substances. Just which of these products are the chief culprits is far from being determined, although much work has been done upon the subject. The best known products of fermentation and putrefaction are the following:

Acids.—Milk frequently contains lactic, butyric, acetic, and other organic acids, which result from the common fermentative changes. The higher volatile fatty acids come especially from the spore-bearing anaerobes, and result from putrefactive decomposition in the milk. All these acids are irritants, by virtue of their acid properties. If present in considerable concentration in a healthy digestive tract or in a more moderate concentration in a person with an irritable stomach or with deranged digestion they may be factors in exciting vomiting or diarrhea. It is probable that when these acids produce acute symptoms they result more from fermentative processes within the gastro-intestinal tract rather than from those produced in milk before it is taken.

The presence of excessive amounts of acids in the intestinal tract may indirectly produce chronic poisonous conditions by robbing the organism of alkali.

Basic substances.—The true bacterial toxines were first thought by chemists to be basic substances resembling alkaloids. We now know that this is not the case. Many basic substances, some of them poisonous, have been described as ptomaines; but their relation to the poisonous properties of milk is very doubtful. Tyrotoxicon, one of the chief of these, studied by Vaughan, is now admitted to be rare in milk and cheese and its chemical composition undetermined. The ptomaines contain nitrogen and have generally been looked upon as products of decomposition of the proteid substances in milk.

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[a] Kamen: Zur Etiologie der Gasphlegmone. Cent. f. Bakt., Orig., vol. 35, 1904, pp. 555, 686.

Cholin is a base which can readily be split off from the fatty body lecithin. Milk contains about one-tenth of 1 per cent of lecithin. While cholin itself is not very poisonous, Hunt has shown that acetylcholin is 100,000 times more toxic than cholin itself and that there are other poisonous cholin compounds. While acetylcholin has never been demonstrated in milk, theoretically it is possible to have a small quantity of this and allied poisons formed. Lecithin may be decomposed by bacterial action, and it is not unlikely that a similar action is responsible for poisonous bodies of this group.

Sulphur compounds.—Sulphur compounds, such as hydrogen sulphide, while poisonous, are not present in sufficient quantity in milk to give serious concern.

Aromatic products, such as the phenols and cresols, skatol, indol, etc., result from the putrefaction of albumins of the common proteid foods. Milk yields considerable quantities of tyrosin from its casein. The phenols and cresols are derived from the breaking down of the tyrosin. Herter thinks that the phenols can not be regarded as important toxic agents; moreover, they are produced by putrefactive processes in the intestinal tract and are not contained in any quantity in milk when it is consumed.

To sum up our knowledge, we find that certain bacteria contained in milk, such as tubercle, cholera, dysentery, typhoid, diphtheria, etc., may induce specific diseases. Other organisms, such as the virulent streptococci and staphylococci, are capable of causing sever inflammations of the gastrointestinal tract. The spore-bearing organisms set up putrefactive and proteolytic changes, and may produce poisons. This occurs in milk, both within and without the body. The nature of these poisons is not known. So far as we know, the true bacterial toxines play little if any rôle in milk poisoning.

INFANT FEEDING.

Prepare cow's milk as we may, we can not shut our eyes to the fact that it is out of the question to anticipate such good results from artificial feeding as from breast feeding. It is well known that the lowest death rate for the first year of life is shown among those infants who are fed on good human breast milk.

It is the milk of other animals, usually the cow, which directly or indirectly kills the greatest number of infants. All are agreed that if a child must be artificially fed it is best to use fresh, pure milk; but when we consider that thousands of infants in our large cities must depend upon the milk of a cow many miles away, we are confronted with a difficulty not readily overcome. Nature did not intend the young of one species to be raised upon the milk of another, much less did it intend that milk to be dirty, stale, and bacteria-laden. We have unanimous testimony that such milk, especially in the heated months of summer, is the cause directly or indirectly of the excessive infant morbidity and mortality.

The average city market milk that has already deteriorated in quality can not be revivified. No known process will make bad milk good milk; but further fermentation and putrefaction in the milk can be stopped, and pathogenic organisms killed, by heating it to 60° C. for twenty minutes. Bad milk, whether heated or unheated, is unfit for infant feeding, but if infants must depend upon old dirty and uncared for milk it would be much better, especially in the summer months, to practice pasteurization, in spite of its alleged disadvantages.

The quantity of certified or clean milk in any community is but a drop in the bucket, and until health officers can assure a good quality of milk the only protection we have is the expedient of heating it.

It is by no means claimed that heated milk is the ideal to be attained. On the contrary, we want good, fresh milk that needs no heating. At present it is exceedingly difficult to obtain such milk in our large cities, and anyone who investigates the matter carefully will soon convince himself that it will be many years before this is possible and only after a revolution of the milk industry. In the meantime we must protect ourselves.

Physicians who have had large experience in the care and feeding of infants have a prejudice against the use of heated milk for prolonged periods. While it is admitted that the use of heated milk greatly diminishes the amount and seriousness of infantile diarrheas, it has been stated that while the children at first do well they may become flabby and anemic and the subjects of scurvy. It is probably not the heating but some other factor in the milk that induces scurvy.

We have the published testimony of a large number of physicians to the effect that the use of pasteurized milk produces no harmful effects that may be attributed to the heating. But when all is said and done the pasteurization of milk for infant feeding can neither be recommended nor discountenanced as a general proposition. The saying that "one man's meat is another man's poison" applies with special significance to the artificial feeding of infants. The general pasteurization of all milk used for the nourishing of infants would be as irrational as the general use of one formula. Each infant is a law unto itself, and whether it is to receive heated or unheated milk must depend entirely upon the conditions, especially the season of the year and the quality of the milk available.

Scurvy.—Scurvy occurs in children fed both upon pasteurized and unpasteurized milk; it may even occur in breast-fed infants. Scurvy is at most a comparatively rare disease. As there are countries where, despite sterilization, scurvy practically never occurs, the cooking of the milk can not be the only cause of this disease. It is not a new disease, but was described in infants for the first time only a decade ago. Even at the present time the disease is often not recognized by clinicians. Formerly the condition was called "acute ricketts" (Moeller). In Germany we are told the disease is either exceedingly rare or not recognized. For a long time the French claimed that the disease did not exist among them, but during the past two or three years there have been occasional reports of isolated cases (Netter).

The disease was first studied by English clinicians and we are especially indebted to Barlow, who, after a study of 11 cases with postmortem results of 2, showed the essential features of the disease and gave it the name of scurvy. It is often spoken of as Barlow's disease,[a] or the Moeller[b]-Barlow disease.

We do not know whether scurvy has increased greatly during the past twenty years, or whether our more precise knowledge of the disease has made this apparent. Those who believe the disease is increasing attribute this fact to the use of dried proprietary infant foods and the increasing use of heated milk.

The proper treatment of infantile scurvy gives almost miraculous results. "Within a few hours a pitiable, suffering little paralytic is transformed to a contented baby waving its arms and legs in the sheer joy of living." This may be simply brought about by the use of fresh milk, fruit juices (orange, grape, or pineapple), beef juice, egg albumen, or puree of potato, according to the child's digestive capacity. Scurvy is thus not only readily preventable, but amenable to treatment, and it would seem that those who have to choose between the use of badly contaminated milk, with its serious consequences, and the remote possibility of scurvy as a result of pasteurization, should not hesitate long in the choice.

I have made a careful compilation from the literature of the results of raising children upon heated milk, and find hundreds of instances recorded, especially by French observers, to the effect that children flourish well upon heated cow's milk and without the production of scurvy. But in view of the fact that scurvy is either rare or not recognized in France we must examine these figures critically. Some of this evidence follows:

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[a] Barlow: Med. and Chir. Transactions, London. Vol. 66, p. 83.

[b] Moeller: Akute Rachitis. Königsberg. med. Jahrb., Bd. I (59), and Bd. III (62).

Variot[a] in a recent communication sums up his experience with the use of heated milk for infant feeding as follows:

> At the dispensary of La Goutte de Lait de Belleville, which I have directed since 1892, we have distributed for twelve years in the poorest quarters of Paris about 400,000 bottles of sterilized milk to more than 3,000 infants of the working class deprived of their mother's milk. With my collaborateurs, MM. Drs. Dufestel, Lazard, and Roger, we have made a study of the artificial feeding with sterilized milk and the results of our experiments are so decisive, each case controlled by weight, and an examination of the organs and functions, that we think our results merit publication.
>
> The milk received from farmers in the country is heated to 108° C. before transportation in the bottles of half a liter, stoppered with cork and the medical seal. This milk keeps several days without alteration, even during the greatest heat of summer. It is delivered daily at the Belleville dispensary to 100–150 infants. Every week or oftener if necessary the infants are weighed and inspected with care, records of which are kept. The following are some of the conclusions of the results of twelve years' experience:
>
> 1. The milk sterilized at 108° C. preserves all of its nutritive value. It is not inferior to milk pasteurized at 80° C. or with heating at 100° C. in the apparatus of Soxhlet.
>
> 2. The destruction by the heating of the enzymes, the slight alteration of the lactose, the doubtful precipitation of the citrate of calcium, or the alteration of the lecithins does not affect its assimilability in an appreciable manner. Not one case of infantile scurvy has been observed by the dispensary.
>
> 3. Thanks to this sterilized milk we have been able to raise not only healthy infants, but also atrophic infants, retarded in their development as a result of gastro-intestinal troubles.
>
> 4. Rachitis did not develop in any of the infants.
>
> 5. In 3,000 infants of the poorest class about 3 or 4 per cent showed themselves incapable of using sterilized milk.
>
> 6. Constipation and anemia were not rare among the infants raised by this method. On the other hand the summer diarrheas were markedly attenuated in severity.

Berlioz[b] reports favorable results from the use of sterilized milk. He believes that with such milk we are capable of enormously reducing infant mortality. From 1894 to 1897 he distributed sterilized milk to the poor of Grenoble during the months of July, August, and September. It was sterilized in an autoclave at 110° C. for half an hour in bottles containing 200–250 cubic centimeters.

[a] Variot, M. G.: Valeur nutritive du lait de vache stérilisé à 108° pour l'allaitement artificiel. Comp. rend. des séances de l'Acad. d. Sci., vol. 139, 1904, p. 1002.

[b] Budin, M. P.: Sur le lait stérilisé. Bull. de l'Acad. de méd., 3^{me} sér., vol 37, 1897, p. 685.

The use of this milk gave the following death rates:

Year.	(A.) Children fed on milk not sterilized (per 1,000).	(B.) Children fed on sterilized milk (per 1,000).
1894	66.8	25.6
1895	86.9	42.2
1896	54.0	16.1
Average	69.3	27.9

The difference in favor of sterilized milk is much more striking than the figures indicate, for Class A includes bottle and breast-fed children, while Class B includes only bottle-fed children. Further, the first figures are compiled from children of the better class, while the latter are drawn from children of the poorer classes.

Carel,[a] from observations upon infants of the working classes in Paris, recommends the use of sterlized milk from the time of weaning. He believes further that the use of sterilized milk has brought about a reduction in the dangers to infants to a minimum. In infants of normal weight and good health, nourished with sterilized milk, the dentition proceeds normally and the mortality from gastroenteritis is nil.

From a comparison of two series of observations of infants coming from families of the same social conditions, living in the same quarter, and of whom the mothers had received the same advice, there occurred 31.8 per cent of rachitis among those nourished with ordinary milk (210 observations). The proportion of rachitis in 373 infants who received sterilized milk was only 15 per cent. None of the 373 infants given sterilized milk presented any symptoms of infantile scurvy (Barlow's disease).

Budin and Chavane,[b] 1894, reported 15 successful cases in 1892 and 1893 of infants fed upon milk sterilized at 100° C. in a water bath and used within twenty four hours. They give in detail the increase in weight and the condition of each infant.

Maygrier,[c] 1901, states that of 590 infants who received sterilized milk from 1878 to 1901 not one died of diarrhea. Much similar testimony to the same effect could be brought forward.

While the evidence is clear that many children are successfully raised upon milk heated even above the boiling point, on the other hand we have a number of cases of scurvy following the use of heated milk, the condition ceasing with the use of raw milk. Of the 379 cases of scurvy brought together in the report of the American Pediatric Society in 1898, sterlized milk was the previous diet of 107.

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[a] Carel, Armand. Le lait sterlisé. Paris, thèses, 1902-3.

[b] Budin, P., and Chavane, A. De l'emploi, pour les nourrissons, du lait stérilisé à 100 degrés au bain-marie. Bull. de l'Acad. méd., 3me sér., vol. 32, 1894, p. 67.

[c] Maygrier. La consultation de nourrissons à la Charité, de 1898 à 1901. Obstétrique, vol. 6, 1901.

Every physician knows from observation that some children do very well upon cooked milk. It is also generally known that it is often only necessary to correct the general dietary, to prevent overfeeding, and to correct the formula, in order to convert an apparently bad milk which is not agreeing with an infant into a good food. Often at the same time the heating of the milk is discontinued and the good results of the change are credited to the use of raw milk.

The results of animal experiments are somewhat contradictory and rather unsatisfactory. Observation upon infants, however, gives us definite results. Finkelstein, for instance, has shown that infants evidently do worse with cooked woman's milk than with raw milk. These experiments correspond entirely with similar experiments made with cow's milk upon calves. Finkelstein next made the experiment of feeding cooked and uncooked cow's milk to children. He used the best milk obtainable in Berlin, and was careful to use the same milk in both cases. The additions, dilutions, and other conditions were precisely the same. The only factor which varied was that in one instance the milk was cooked and the other raw. A study of these parallel cases does not show any essential difference so far as nutrition is concerned between those receiving the raw milk and those receiving the cooked milk. Finkelstein tells us that similar experiments made in Stockholm, but continued over a longer time, viz, three years, confirms his observations and failed to show any difference between the two methods.

So far as other metabolism experiments on infants are concerned they likewise practically all point to the conclusion that raw milk has no advantage over cooked milk. This is especially evident with respect to the organic constituents of milk. So far as the metabolism of the mineral substances is concerned the evidence is somewhat contradictory. Thus, Mueller and Cronheim found the calcium balance to favor raw milk.[a] These results have not been confirmed by the work of others.

Krasnogorky found that iron is taken up more readily from cooked than from raw milk.

So far as we are able to conclude from the evidence at hand upon metabolism experiments, raw milk certainly has no advantages over cooked milk.

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[a] Finkelstein, H.: Die rohe Milch in der Säuglingsernährung. Therap. Monatsh., vol. 21, October, 1907, p. 508.

When we consider that we know practically nothing of the essential nature of scurvy we must be cautious in considering the connection between pasteurization and scurvy as cause and effect. Rotch,[a] for instance, says:

> In those cases where scorbutus has apparently occurred in infants who were being fed on milk heated to 212°, it may have been some other quality in the milk which produced the scorbutus, and that either the percentages which the infant has been fed upon are not those which are adapted to and fitted to that especial infant or that it is an exceedingly dirty milk which they have been boiling at 212°, and which necessarily does not become a sterile milk in the meaning of infecting the individual.

The unsatisfactory state of our knowledge upon this subject is evident from the following views recently expressed:

Rummell[b] doubts the relationship between the Mueller-Barlow's disease and sterilized milk. The cause of this disease, despite the great literature upon it, is entirely unknown. The fact that the occurrence of infantile scurvy varies so much in different regions leads one to suppose that perhaps it has some relation to the food of the cow rather than to the heating of the milk. That the disease seems to be brought about sometimes by high-grade sterilization of the milk, in an analogous way to scurvy in adults, seems probable. Animal experiments have been very contradictory and have not yet done much to clear up the situation.

Koeppen[c] looks upon scurvy as an auto-intoxication brought about by intestinal putrefaction, which process is favored in children artificially raised.

Recent evidence (see Schereschewsky's paper, article No. 23, in this bulletin, p. 687) points to the fact that scurvy may be brought about by lack of the vegetable inorganic salts of alkaline bases, especially potassium, in the infant's dietary. This, combined with the injurious effects of a high percentage of fat in the food, may bring about serious disturbances of digestion and metabolism, favoring the production of the scorbutic condition. If this view is correct it entirely eliminates the heating of the milk as an etiologic factor.

The admirable work of Holst and Frölich[d] (1907) goes far to clear up many of the doubts concerning the etiology of scurvy. These investigators have produced a disease in guinea pigs practically identical with human scurvy. This was done with a one-sided diet consisting of various sorts of grain, groats, and bread. The guinea pigs did not get the disease when fed upon a one-sided diet consisting of fresh cabbage or fresh potatoes, whereas it was produced by dry potatoes; that is, the disease originates in guinea pigs as well as in man as a result of a diet confined to some special nutriments.

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[a] Rotch, Thomas Morgan: "The pasteurization of milk for public sale." Am. Journ. Pub. Hyg., vol. 17, May, 1907, p. 181.

[b] Rummell, O.: Sterilisierte Milch? Deut. Praxis, vol. 13, 1904, p. 201–207.

[c] Koeppen: Zur Moeller-Barlow'schen Krankheit. Jahrb. f. Kinderheilk. Bd. 44 1897.

[d] Holst, A., and Frölich, T.: "Experimental studies relating to ship beriberi and scurvy." (II) "On the etiology of scurvy." Journ. Hyg., vol. 7, Oct., 1907, p. 634.

Holst and Frölich also observed that the disease in guinea pigs is favorably influenced by different sorts of nutriments known from human experience as "antiscorbutics." They found, however, that at least one of their nutriments, viz, cabbage, loses a deal but not all of its preventive power when boiled for half an hour at 110° C. There is no evidence to show that moderate heating, such as is used in the pasteurization of milk, in any way affects the scorbutic or antiscorbutic qualities of a food.

Infant mortality.—It is now well established that the large majority of infantile deaths is caused by gastro-intestinal diseases. Further, that this great fatality occurs especially among artificially raised infants, and finally that the vast majority of cases and deaths from bowel troubles in children occur during the heated term. The infant mortality in all countries is shockingly high. This is shown to be unnecessary by the fact that infants who are well cared for show a relatively low mortality. Defective feeding is the active cause of this high mortality, while heat, humidity, and bad surroundings are contributary causes. It must be remembered that the normal intestinal mucous membranes are permeable to bacteria, and more so during the period of infantile than of later life. Hence one of the great dangers of using bacteria-laden milk. While the factors involved in this "slaughter of the innocents" are numerous, primarily or secondarily they depend upon the activity of micro-organisms. Freeman[a] believes that the decline in the infant mortality in the United States during the last ten years, and especially in New York City, is due for the most part to the decline in mortality from summer diarrhea, and states "that the general adoption of pasteurized and sterilized milk for infant feeding is by far the most important agency." A definite example of the diminution in mortality from pasteurizing the milk occurred in the infants' hospital at Randalls Island, where the mortality in 1897, with raw milk, was 44.36 per cent, while in 1898, with pasteurization of the milk, it was 19.80 per cent.

Numerous similar instances of the beneficial effect upon infant mortality and morbidity are found in the literature.

A reduction in the infant mortality may be accomplished without the heating of the milk. This has been shown by Doctor Goler, who conducted an aggressive campaign to improve the milk supply for infant feeding in Rochester, N. Y. His methods consisted mainly in education in the nursery and on the dairy farm. The clean milk obtained thus and distributed through milk depots resulted in lowering the death rate in children under 5 years from 33 per cent from all causes to 20 per cent, and now (1907) it is 15 per cent.

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[a] Freeman, Roland G.: Medical News, Sept. 5, 1905.

Park and Holt[9] studied groups of infants in the tenement houses and institutions in New York for periods of about three months in the summers of two years (1902-3). This work is the most important evidence we have on the subject, for it combines careful clinical observation with laboratory studies. Although the number of cases was comparatively small, the results obtained were almost identical during the two summers, and indicate that even fairly pure milk, when given raw in hot weather, causes illness in a much larger percentage of cases than the same milk given after pasteurization. A considerable percentage of infants, however, did apparently as well on raw as on pasteurized milk. Park and Holt conclude in part:

> The number of bacteria which may accumulate before milk becomes noticeably harmful to the average infant in summer differs with the nature of the bacteria present, the age of the milk, and the temperature at which it has been kept. When milk is taken raw the fewer bacteria present the better are the results. Of the usual varieties, over 1,000,000 bacteria per cubic centimeter are certainly deleterious to the average infant. However, many infants take such milk without apparently harmful results. Heat above 170° F. (77° C.) not only destroys most of the bacteria present, but apparently some of their poisonous products. No harm from the bacteria previously existing in recently heated milk was noticed in these observations, unless they had amounted to many millions, but in such numbers they were decidedly deleterious.
>
> When milk of average quality was fed sterilized and raw, those infants who received milk previously heated did on the average much better in warm weather than those who received it raw. The difference was so quickly manifest and so marked that there could be no mistaking the meaning of the results.
>
> A few cases of acute indigestion were seen immediately following the use of pasteurized milk more than 36 hours old. Samples of such milk were found to contain more than 100,000,000 bacteria per cubic centimeter, mostly spore-bearing varieties. The deleterious effects, though striking, were not serious or lasting.
>
> After the first twelve months of life, infants are less and less affected by the bacteria in milk derived from healthy cattle. According to these observations, when the milk had been kept cool the bacteria did not appear to injure the children over 3 years of age at any season of the year, unless in very great excess.

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[9] Park, Wm. H., and Holt, L. Emmett: "Report upon the results with different kinds of pure and impure milk in infant feeding in tenement houses and institutions of New York City; A clinical and bacteriological study." Medical News, vol. 83, 1903, p. 1066.

The general practice of heating milk, which has now become a custom among the tenement population of New York, is undoubtedly a large factor in the lessened infant mortality during the hot months.

Only the purest milk should be taken raw, especially in summer.

No discussion of the subject is complete without recognition of the debt the world owes Mr. Nathan Straus for his early and persistent advocacy of pasteurization and the establishment of his infants' milk depots. Through his influence and philanthropy this movement has now spread to many cities of this country and abroad.

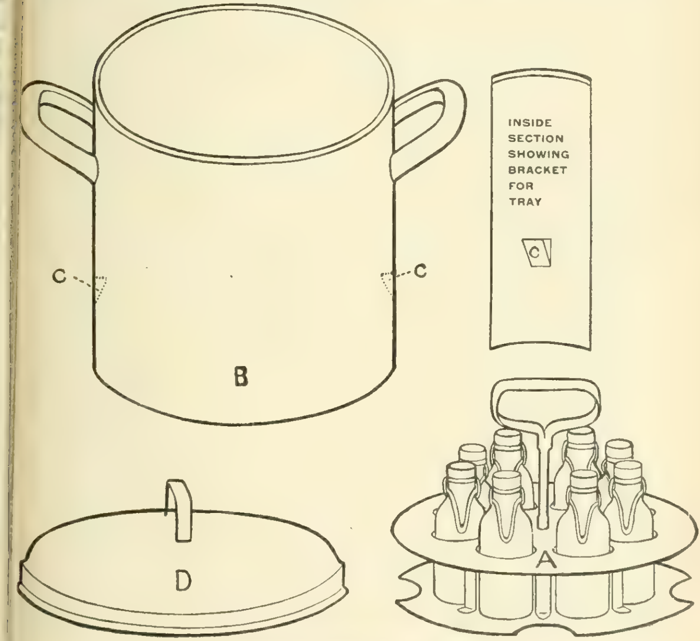

FIG. 64.—Home pasteurizer. (See p. 675

Mr. Emile Berliner, of Washington, has also for many years pointed out the dangers in raw milk and taught the wisdom of "scalding" milk.

HOME PASTEURIZATION.

If pasteurization is to be done perhaps the best place to do it is in the home, but the heating of milk to just 60° and the holding of it to just that temperature for twenty minutes, then cooling it rapidly, requires intelligence and careful manipulation. With the possible exception of infant feeding, it would perhaps be better and cheaper to pasteurize the milk in bulk under competent supervision instead of leaving it to the usual carelessness of cooks, who can not be expected to master the technic nor appreciate the difficulties. Imperfect pasteurization may be worse than none, for it may result only in further contamination of the milk.

Milk pasteurized in the home is commonly heated too high and not rapidly cooled.

The most practical home pasteurizer is that devised by Freeman.[a]

The following experiments, made in the Hygiene Laboratory, with Freeman's pasteurizer show its efficiency:

TEST NO. A WITH FREEMAN'S PASTEURIZER.

Temperature of milk, 9° C.

Temperature of water in jacket, 25° C.

Milk introduced into boiling water and removed from the fire.

Milk temperature—	° C.
Five minutes after immersion in boiling water	47. 5
Ten minutes after immersion in boiling water	63
Fifteen minutes after immersion in boiling water	67. 4
Twenty minutes after immersion in boiling water	68. 8
Twenty-five minutes after immersion in boiling water	68. 9
Twenty-eight minutes after immersion in boiling water	68. 9

Whole time, twenty-eight minutes.

Above 67° C. for thirteen minutes.

Took fifteen minutes in running tap water, at 22° C., to cool milk to 30° C.

The results follow:

	Bacteria per cubic centimeter.[b]	
	Raw milk.	After pasteurization.
Milk from—		
Dairy I	34, 600, 000	300
Dairy K	1, 050, 000	50
Dairy L	80, 000	400
Dairy M	2, 200, 000	200
Dairy N	2, 100, 000	1, 200
Dairy O	1, 900, 000	None.
Dairy P	2, 400, 000	600
Dairy Q	2, 000, 000	150

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[a] Freeman, Rowland G.: "Low temperature pasteurization of milk at about 68° C. (155° F.)." Arch. of Ped., 1896.

[b] Colonies on agar plates after twenty-four hours' incubation at 37° C.

TEST No. 3 WITH FREEMAN'S PASTEURIZER.

Temperature of milk, 11° C.

Temperature of water in jacket, 22° C.

Temperature of milk—	° C.
Five minutes after immersion in boiling water	50
Ten minutes after immersion in boiling water	63
Fifteen minutes after immersion in boiling water	66.6
Twenty minutes after immersion in boiling water	67.5
Twenty-five minutes after immersion in boiling water	67.7
Thirty minutes after immersion in boiling water	67.4
Thirty-five minutes after immersion in boiling water	67
Forty minutes after immersion in boiling water	66.6
Forty-five minutes after immersion in boiling water	66

It took thirteen minutes in running tap water, at 22° C., to cool the milk to 30° C.

Whole time, forty-five minutes.

Above 65° C., thirty minutes.

	Bacteria per cubic centimeter.[a]	
	Raw milk.	After pasteurization.
Milk from—		
Dairy A	1,900,000	300
Dairy B	2,500,000	500
Dairy C	2,100,000	50
Dairy D	440,000	None.
Dairy E	1,090,000	200
Dairy F	29,800,000	1,750
Dairy G	1,420,000	None.
Dairy H	590,000	2,650

[a] Colonies on agar plates after twenty-four hours' incubation at 37° C.

NOTE.—Recently (November, 1907) Freeman has modified his pasteurizer so that the milk is heated to 60° C. for forty minutes. (See his article on "The ferments in milk and their relation to pasteurization," in the Jour. of the Amer. Med. Assn., Nov. 23, 1907, Vol. XLIX, No. 21, p. 1740.)

Milk is frequently pasteurized by simply placing the bottle of milk as it is received in a pot of water, the water boiled for a variable length of time, and then cooled. As will be shown by the following experiments, this is not always an entirely safe procedure for the purposes of home pasteurization. The depth of water in which the bottle is immersed markedly affects the results. The neck of the bottle must always project above the water, and unless the pot has a lid the upper layers of the milk may escape heating, especially if the contents have not been well shaken up and the thick cream, which is in part turned to butter as a result of agitation on the delivery wagon, prevents circulation of the fluid.

It will be seen in some of the experiments made by myself in the Hygienic Laboratory that, contrary to what might be expected from the physics of fluids, the top layers of the milk are sometimes not as hot as the bottom or require a much longer time to heat up.

EXPERIMENT No. 1.

Pint mixed market milk.

Bottle immersed in water to its lip.

Distinct cream line from standing over six hours before heating; thick cream, almost butter, floating on top.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
26	26.5	25.5	....	(a)	
30	26.5	26.5	2	(a)	
40	27	28.75	4¼	(a)	
50	30	32	7	(a)	
60	35	39	9½	(a)	
70	41	45	12	(a)	
75	45	49	13	(a)	
80	50	53.5	14½	(a)	
83	....	....	....	....	Simmering.
85	55	58	15½	(a)	
87	58	60.5	16¼	(b)	
88	60	62.5	16¾	(b)	
91	63	66	17½	(b)	
92.5	65	68	18¼	(b)	
97	70	73	19½	(c)	
98	71	75	20	(c)	Boiling.
100	75	80	21¼	(c)	
100	78	83	23	(c)	
100	80	85	23½	(d)	

[a] Innumerable.

[b] Somewhat less innumerable.

[c] Distinctly less innumerable.

[d] About 200,000.

EXPERIMENT No. 2.

Pint mixed market milk.

Bottle immersed in water 6 inches, 1 inch out.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
28	14	....	....	9,000	
60	32	....	5	....	
70	40	....	7	....	
80	48	....	9	....	
85	50	....	10	8,000	
90	55	....	11	4,000	
94	60	....	12	750	
98	65	....	12½	600	
100	70	....	13	80	

EXPERIMENT NO. 3.

Pint mixed market milk.
Bottle immersed in water 6 inches, 1 inch out.
Distinct cream line from standing twenty-four hours.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
23	20	13	....	4,000	
30	21	21	2⅓	....	
40	23	24.5	3¼	6,000	
50	28	30	6¼	....	
60	35	36.5	8¼	....	
70	44.5	44.5	9⅓	....	
80	53	53	13	....	
81.5	55	54.5	13	9,000	
85.	60	58	13⅔	6,000	Simmering.
88	61.5	59	14	....	Scum.
89	63.5	60.5	14½	7,500	
90	65	62	14¾	....	
92	67	64	15¼	600	
95	70	68	16	1,500	Boiling.
96	75	73	18	900	
....	77	76	19	([a])	Scum.

[a] Fewer, but numerous.

EXPERIMENT NO. 4.

Pint mixed market milk.
Bottle immersed in water 4½ inches, 2½ inches out.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
22	21	15	....	4,300	
30	22	17	1¾	....	
40	22.5	20	4¾	....	
50	24	23	4	....	
60	27	25.5	5⅓	4,500	
70	31	32	6¼	....	
80	35	40.5	9	....	
85	36	45	10	....	Simmering.
93	44	54	11¾	6,000	
96	47	58	12½	....	Boiling.
....	50	59	13	3,000	
....	55	61	13¼	3,200	
....	58	62	13½	3,500	
....	60	64	13⅔	....	
....	65	65	13¾	4,000	
....	70	67	14¼	2,000	
....	73	68.5	15	3,100	

EXPERIMENT NO. 5.

Pint mixed market milk.
Bottle immersed in water 4 inches, 3 inches out.
Recently mixed, no distinct cream line.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	*Minutes.*		
30	23	16	....	480	
40	22	20	1½	....	
50	24	23	3	....	
60	25	27	5	....	
70	27	33	6½	....	
80	29	37	8	812	
86	32	45	9	....	Simmering.
90	33	48	9½	....	
91	36	51	10¼	....	
98	40	57	10¾	....	Boiling.
....	50	65	12½	760	
....	55	70	13½	150	
....	58	74	14½	58	
....	60	75	15	35	
....	63	80	16½	0	
....	65	81	17	1	
....	70	85	19½	1	

EXPERIMENT NO. 6.

Pint mixed market milk.
Bottle immersed in water 3 inches, 4 inches out.
No cream line.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	*Minutes.*		
24	13	....	....	1,500	At start.
40	17	....	3	....	
50	20	....	4	....	
60	24	20	5½	....	
70	29	24	6½	....	
80	35	30	8	....	
87	....	....	9	....	Water simmering.
90	44	38	9½	....	
95	....	....	10½	....	
100	55	49	11½	1,800	Water boiling.
100	60	54	12½	1,700	
100	65	61	13½	230	
100	68	64	14½	..	Scum 1,300.
100	70	67	15½	55	
100	72	69	16	..	Scum 1,500.
100	75	73	17	6	
....	77	....	....	..	Scum 2,000.

EXPERIMENT No. 7.

Pint mixed market milk.
Bottle immersed in water 3 inches, 4 inches out.
Partial cream line.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
26	16	17	....	2,000	
30	19	20	1	....	
40	20	21	2	....	
50	22	22.5	3¼	....	
60	27	25.5	4¾	1,000	
70	33	30	6¼	....	
80	40	36	7⅔	....	
88	....	....	9	....	Simmering.
90	49	43.5	9¼	....	
97	57	51	10⅓	....	Boiling.
....	60	53	11¼	1,000	
....	63	55	11½	350	
....	66	57.5	12	60	
....	70	64	13¼	9	
....	75	70	14⅔	5	Some scum.
....	78	75	16	2	Scum.

EXPERIMENT No. 8.

Pint mixed market milk.
Bottle immersed in water 5 inches, 2 inches out.
Cream line; mixed a little, not much, with pipette.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
25	19	14.5	....	215	
40	23	22	3	....	
50	28	28	6¼	230	
60	32	35.5	8⅓	....	
70	40	43	10⅓	....	
80	49	52.5	13	....	
83	52	56	14	....	
86	55	59.5	15	2,000	
91	60	65	16¼	2,100	
94	63	68.5	17	(a)	
95	65	71	17½	(a)	
97	68	72.5	18	....	Water boiling.
100	69	75	19	[a]	Scum.
100	72	78	19½	410	
100	74	80	20	......	Do.

[a] Lost.

Experiment No. 9.

Quart mixed market milk.
Bottle immersed in water to lip.
Distinct cream line from standing at least five hours.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
24	25	22	....	450,000	
30	25	24	2¼	....	
40	26	25	5	....	
50	28	30	8¼	....	
60	35	32	12	550,000	
70	45	43.5	15	550,000	
80	52	52.5	18½	....	
82	55	57	19½	550,000	
84	58	57.5	21	550,000	
85	60	57.5	21¾	550,000	
87	....	....	....	....	Simmered.
88	63	62	22¾	425,000	
90	65	64	23	175,000	
97	69.5	71.2	25	13,000	Boiling.
100	75	79.5	28½	33	

Experiment No. 10.

Quart mixed market milk.
Bottle immersed in water to lip.
Recently mixed, no distinct cream line.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
27	24	20	....	27	
30	24.5	20	1	.....	
40	29.5	21	3¼	....	
48	35	....	5¾	296	
50	36	27	6	....	
60	44	32	8½	....	
65	45	33	9½	208	
69	50	37	11	1,400	
70	51.5	37.5	11¾	....	
74	55	39	12¼	874	
76	58	41	12¾	3,180	
78	60	43	13¼	1,930	
80	62	45	14	....	
81	63	46	14¼	1,300	Simmering.
83	65	47.5	15	370	
88	70	53	16	115	
90	72	56	16¾	....	
95	75	61	17¾	59	
100	80	65.5	20	.. ..	Boiling.

Experiment No. 11.

Quart mixed market milk.

Bottle immersed in water 5 inches, 4 inches out.

No distinct cream line.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
28	23	16	....	7,100	
40	23.5	20	2¼	.....	
50	24	23	4½	.....	
60	24.5	28	6¼	....	
70	25	32	8½	....	
80	30	40	10½	12,000	
85	......	....	......	......	Simmering.
90	38	51	13½	....	
97	41	56	15	....	Boiling.
....	50	64	16	9,500	
....	55	67	18	7,400	
....	58	71	19¼	5,400	
....	60	72	20	..	
....	61	74	20½	2,200	
....	65	75	21	......	
....	68	77	21½	3,600	
....	70	78	22	1,700	

Experiment No. 12.

Quart mixed market milk.

Bottle immersed in water 5 inches, 4 inches out.

Distinct cream line.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
24	18	14	....	26,000	
30	18.2	....	2½		
40	19	20	3½	30,000	
50	20	26	5½		
60	23.5	30	7½	.	
70	31	35	9½	.	
80	37	41	11½	.	
84	39	44	12		Simmering.
90	45	50	14		
95	...	.	15		Boiling.
..	58	59	16	30,000	
.	61	[illegible]	16	21,000	
	[illegible]	[illegible]	16	[illegible]	
....	68	62.5	17	33,000	
...	70	63	17½	18,000	
.....	72	64	17	[illegible]	
..	71	65	18	9,000	
......	75	[illegible]	18	9,000	
.	80	72	20		

EXPERIMENT No. 13.

Quart mixed market milk.

Bottle immersed in water 4 inches, 5 inches out.

Cream line very distinct from long standing.

The bottom milk hotter than the top, probably on account of heavy viscid cream on top that did not circulate.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes		
20	20	12	....	[a] 4,000	Control.
30	20	14	5	....	
40	20.5	16	5¼	....	
50	21	20	6¾	....	
60	21.5	24	8½	....	
70	22	28	10¼	....	
80	23	36	12¼	....	
86	26	41	13½	....	Simmering,
90	28.5	43	13¾	....	
95	35	47	15¼	....	Boiling,
100	44	55	17	(b)	
100	49	60	18¼	[a] 4,000	
100	50	61	19½	....	
100	54	65	20	(b)	
100	60	70	22½	(b)	
100	63	72	23	340	
100	65	74	24	150	
100	70	79	26¾	8	
100	73	82	28	.....	
100	75	83	30	13	

[a] About.

[b] Lost.

EXPERIMENT No. 14.

Quart mixed market milk.

Bottle immersed in water 4 inches, 5 inches out.

No cream line.

Water temperature.	Milk temperature.		Time.	Colonies per loop in agar.	Remarks.
	Top.	Bottom.			
°C.	°C.	°C.	Minutes.		
22	13	....		5,760	
30	15	....	2		
40	18	....	4	....	
50	22	....	5	....	
60	27	..	8	....	
70	32	...	8½	..	
80	39	..	10	....	
90	47		12	.	
95	50		13	....	
			14		Boiling.
100	55		14	8,000	
100	60	......	15	14,000	
100	65	..	16	4,000	At 66°, scum
100	70		18	380	
100	73		19	....	Scum.

Nathan Straus has modified the Freeman pasteurizer by leaving off the cups, thus making it simpler and cheaper. He gives the following directions for the manufacture of this home pasteurizer:

	Size I: Eight 3-ounce bottles.	Size II: Eight 6-ounce bottles.	Size III: Six pint bottles.
Height of pan	$10\frac{1}{8}$ inches	$10\frac{1}{8}$ inches	14, inches.
Diameter of pan	do.	do.	$10\frac{1}{8}$ inches.
Distance of top of bracket from bottom of pan	$3\frac{7}{16}$ inches	$4\frac{5}{8}$ inches	$6\frac{1}{2}$ inches.
Amount of water	5 quarts	$6\frac{3}{4}$ quarts	9 quarts.

The following directions are abstracted from Mr. Straus's instructions for the use of his home pasteurizer:

Emphasis is laid on the fact that only fresh, clean milk, which has been kept cold, should be used.

After the bottles have been thoroughly cleaned they are placed in the tray (A) and filled to the neck. Then put on the corks or patented stoppers without fastening them tightly.

The pot (B) is now placed on the wooden surface of the table or floor and filled to the supports (C) with boiling water.

Place the tray (A) with filled bottles into the pot (B) so that the bottom of the tray rests on the supports (C), and put cover (D) on quickly.

After the bottles have been warmed up by the steam for five minutes, remove the cover quickly, turn the tray so that it drops into the water, replace the cover immediately. This manipulation is to be made as rapidly as possible to avoid loss of heat. Thus it remains for twenty-five minutes.

Now take the tray out of the water and fasten the corks or stoppers airtight. Cool the bottles with cold water and ice as quickly as possible, and keep them at this low temperature until cold.

Use the milk from the bottles and do not pour it into another vessel.

The milk must not be used for children later than twenty-four hours after pasteurization.

COMMERCIAL PASTEURIZATION.

The commercial pasteurization of milk leaves much to be desired, but although it is not always thoroughly carried out, it is by no means a fraud. With a little sanitary supervision on the part of health officers and education on the part of those in charge of the process it may be made efficient.

Commercial pasteurizers are popular with dairymen, not because of the public health aspect, but on account of the economic advantages in improving the keeping qualities of the milk. It is estimated that the expense of a pasteurizer would be paid for in the course of about a year. This estimate is based mainly on the saving of losses from sour milk. The cost of pasteurization is about one-tenth to one half cent a quart.

In order to satisfy public health requirements pasteurizers must be efficient in operation, permitting a definite quantity of milk to be heated to a definite temperature for a definite time (Russell). They must be easy of control, the milk must be heated uniformly throughout, the apparatus must be simple in construction, easily cleaned, economical in use, and arranged to safeguard against reinfection of the milk. Finally, provision must be made for rapid cooling. Given an apparatus of proper construction more depends upon the intelligence and care with which it is run than upon the machine. No pasteurizer is automatic. For instance, I have found that the milk pasteurized in a standard machine contained many more bacteria after the process than before. This was not the fault of the machine, but due to ignorance and uncleanliness.

The following figures show the efficiency of a commercial pasteurizer operated under intelligent though not skilled supervision:

Colonies per cubic centimeter.		Colonies per cubic centimeter.	
Before pasteurization.	After pasteurization.	Before pasteurization.	After pasteurization.
92,000	2,200	380,000	83,000
11[illegible],000	6,000	214,000	87,000
71,000	6,000	6,700	28,200
93,000	6,900	900,000	100,000
105,000	38,000	7,000,000	70,000
1,680,000	80,000	74.000	35,000

The above figures were obtained from a type of machine known as a "Flash pasteurizer," in which the milk is heated momentarily at 73° to 74° C.

The pasteurization of milk is such an important public health measure that it should be under the immediate and constant supervision of the health officer. The milk should be heated a definite temperature for a definite time and then promptly cooled and properly labeled.

RÉSUMÉ ADVANTAGES AND DISADVANTAGES.

Pasteurization saves lives and prevents sickness. Weighing against this great merit we have certain disadvantages connected with the heating of milk. That there are two sides to the question may be judged from the fact that those who have given the matter careful consideration come to diametrically opposite conclusions. From a theoretical standpoint some believe pasteurization to be an unsatisfactory and very feeble way out of a very difficult situation. From a practical standpoint, others find in pasteurization our only practicable safeguard, at least until the general supply consists of good, clean, fresh, safe milk.

One of the chief objections to pasteurization is that it promotes carelessness and discourages the efforts to produce clean milk. It is believed that the general adoption of pasteurization will set back improvements at the source of supply and encourage dirty habits. It will cause the farmers and those who handle the milk to believe that it is unnecessary to be quite so particular, as the dirt that gets into the milk is going to be cooked and made harmless. It is not proposed that pasteurization shall take the place of inspection and improvements in dairy methods. To insure the public a pure and safe milk supply should be regarded as one of the most important duties of the health officer. Whether pasteurization is adopted by a city for its general milk supply or not, no milk should be accepted that does not comply with certain reasonable chemical and bacteriological standards. This would aid the inspectors in enforcing good dairy methods. Pasteurization then must not be used as an excuse to bolster up milk unfit for home consumption. To insure this end, the health officer should have authority to condemn and destroy bad milk, whether or not pasteurization is practiced.

To obtain a good milk supply involves not only an expensive system of inspection and surveillance from the farm to the consumer, but intelligence and a high degree of technical skill on the part of the producer and all others who handle the milk.

We can scarcely conceive of an inspection so thorough and constant as to prevent milk occasionally becoming contaminated with the germs of typhoid, diphtheria, scarlet fever, dysentery, tuberculosis, etc.

If our drinking water is defiled at its source we boil or filter it. It would be much better to prevent its contamination. The same is true of milk. We prefer pure milk, but so long as we can not obtain it we must purify what we get. The situation may well be illustrated by the attitude of an eminent sanitarian in New York, who in his writings and public addresses discourages pasteurization, because theoretically it does not reach the source of the evil, and is not as good in the end as purification of the milk supply through efficient inspection. However, when this same sanitarian is consulted by a large wholesale dealer of New York, who handles many thousands of quarts of more or less old dirty milk a day, he is confronted by a condition, not a theory, and advises pasteurization.

There is a prevalent impression that the pasteurization of milk improves that important article of diet. Heating does not render milk better in any way as a food. All it does is to destroy certain bacteria and some of their toxic products. It checks certain processes of fermentation and putrefaction, thus rendering the milk safer.

On the other hand the evidence seems clear that the pasteurization of milk at 60° C. for twenty minutes does not appreciably deteriorate its quality or lessen its food value.

Pasteurization has been accused of possessing the great disadvantage of inducing scurvy and rickets. It is generally believed that highly heated milk is a contributive factor in the etiology of scurvy. There is certainly no evidence to show that low temperature pasteurization, such as is now recommended, ever in itself induces scurvy. Thousands of children have been raised upon heated milk without the production of this disease, which is comparatively rare, especially in countries such as Germany and France, where the artificial feeding with heated milk is most popular. Scurvy is preventable and amenable to treatment. Rickets results from defective alimentation and improper hygiene and can not be laid at the door of pasteurization.

Comparative observations upon infants under the same conditions show that they flourish quite as well upon heated milk as upon raw milk. Laboratory experiments as well as clinical observations coincide with the view that heated milk is quite as digestible as raw milk. In fact, it is now claimed to be more so. Metabolism experiments indicate that the utilization of calcium and iron in the body is more complete in children fed upon boiled cow's milk than in those fed upon raw cow's milk.

One of the great objections to the pasteurization of milk is that it devitalizes it. If milk contains "life" it has probably lost the last vestige of it after it is from twenty-four to forty-eight hours old and kept under such conditions that it contains myriads of bacteria. It has been shown that heating milk to 60° C. for twenty minutes, while it kills the pathogenic organisms, does not seriously affect the enzymes, and the enzymes are the nearest approach to "life" with which we are familiar in milk. The germicidal properties of milk are not seriously injured at 60° C.

Another objection frequently urged against pasteurization is that some of the bacterial toxins are not killed at the ordinary temperatures used. We do not even know the nature of these poisonous products in milk, much less their thermal death points. The true bacterial toxins are destroyed by heating to a temperature of 60° C. for twenty minutes. It must be remembered that if milk contains bacterial toxins not destroyed by pasteurization it will contain these same poisons if the milk is consumed raw. In fact, the heating of the milk prevents the further formation of such injurious substances.

Pasteurization results in the destruction of the ordinary acid-producing bacteria, nature's danger signal of old milk. The heating interferes with the souring process, so that fermentation of another and perhaps more serious nature may take place without the knowledge of the consumer. It has been shown that certain resistant spore-bearing bacteria have the property of peptonizing the albumens in milk. These bacteria survive the process of pasteurization, and are thus given a free field for growth, whereas in the raw milk these bacteria are largely held in check by the growth of the lactic acid forming organisms. This view started with the work of Flügge and has gradually lost ground for lack of clinical and laboratory confirmation. For instance, Park and Holt found that a few cases of acute indigestion immediately followed the use of pasteurized milk more than thirty-six hours old. Samples of such milk were found to contain more than 100,000,000 bacteria per cubic centimeter, mostly spore-bearing varieties. The deleterious effects, though striking, were not serious or lasting. However, so long as the danger is suspected it makes us cautious to keep pasteurized milk cold and use it promptly.

If it is important for milk to contain lactic acid bacteria. They may readily be added in pure culture after the milk has been pasteurized.

We are told that heating destroys great numbers of bacteria in milk, and thus conceals dirt, but Theobald Smith[a] points out—

> that from a bacteriological standpoint the pasteurization of milk will not conceal dirt, for the reason that the bacteria that come from the udder or the teats will be destroyed, but the bacteria that come from dirt are largely spore-bearing bacteria and these survive. I believe that we could control the quality of milk quite as well after it was pasteurized by bacteriological counts as before, because certain species only would grow or multiply and the indicators would be much better than to-day. If we examine a plate made from milk, for instance, nobody can tell exactly whether the bacteria are due to dirt or whether they are due to the multiplication of ordinary lactic acid bacteria, unless a very careful study of that plate be made. As a rule, if nearly all the colonies are alike we say that they are the result of multiplication; if they are quite different then there has been a good deal of dirt added to the milk. Now it seems to me that with pasteurization it would be possible to control the dirt in milk much better than is done to-day.

Further, it is said that we must not meddle with nature; that pasteurization is an artificial expedient. Nature never intended milk to be collected, transported, and fed to young mammalian animals one or two days after it leaves the mammary gland. Even when fresh, the milk of one species is not well suited to the needs of the young of another species. In the artificial feeding of infants with cow's milk, we are meddling with nature. When artificial feeding is necessary we must endeavor to obtain fresh, pure milk. If this is not possible the milk should be purified, especially in the hot weather. Each infant is a law unto itself.

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[a] Smith, Theobald: Am. Journ. Pub. Health and Journ. Mass. Assn. Bds. Health, vol. 17, 1907, p. 200.

Pasteurization of all of the milk supply of a community may not be desirable. The clean, fresh milk, free from contamination, may not need it. Special cases may require raw milk, but the general public should be protected against the old, dirty, and uncared for milk which forms the bulk of the supply of large cities.

The heating must be done intelligently and under the supervision of the health officer. After heating, the milk is just as liable to serious contamination as before, if not more so. It must therefore be carefully guarded, kept cool, and promptly delivered.

Theobald Smith,[a] 1907, expressed the opinion that pasteurization is the inevitable outcome of the future. He says:

> It seems to me that the real difficulty of the present condition is the transmission of specific disease germs which are not easily controlled by any amount of cleanliness, and these specific disease germs, one and all of them, may be destroyed by the average pasteurization.

Sedgwick[b] voices the opinion of many sanitarians when he states that—

> when all is said and done, I agree with Professor Smith that we have got to pasteurize milk. Cooked milk is the only safe milk and always will remain the only safe milk for the use of mankind. Little by little the idea is spreading that raw milk is apt to be dangerous milk.

Theoretically, pasteurization should not be necessary; practically, we find it forced upon us. The heating of milk has certain disadvantages which must be given consideration, but it effectually prevents much disease and death, especially in infants during the summer months.

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[a] Smith, Th.: Discussion of Rotch's paper on "The pasteurization of milk for public sale." Am. Journ. Pub. Hyg., vol. 17, May, 1907, p. 200.

[b] Sedgwick, W. T.: Discussion of Harrington's paper on "Some of the ways in which infection is disseminated." Journ. Mass. Assn. Bds. Health, vol. 14, Feb., 1904, p. 41.

22. THE THERMAL DEATH POINTS OF PATHOGENIC MICRO-ORGANISMS IN MILK.

THE THERMAL DEATH POINTS OF PATHOGENIC MICRO-ORGANISMS IN MILK.[a]

By Milton J. Rosenau,
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The temperature at which milk should be pasteurized hinges on the thermal death points of the pathogenic micro-organisms which contaminate it. The micro-organisms pathogenic for man most frequently found in market milk are those causing tuberculosis, typhoid fever, diphtheria, scarlet fever, dysentery, and Malta fever. Fortunately none of the organisms causing the above-mentioned acute diseases have resisting spores. Moderate degrees of heat are, therefore, sufficient to render milk safe so far as these dangers are concerned. The streptococci, staphylococci, and most of the bacteria associated with infantile diarrhea are also readily destroyed by heat.

Although it would appear to be a comparatively simple matter to determine precisely the temperature at which micro-organisms die, such work is in fact surrounded by many difficulties and pitfalls; different investigators have come to widely different results. Some of these discrepancies are only apparent and may be explained by the relation of time to temperature. The longer the time of exposure, the lower the temperature necessary to kill any organism. Differences in methods are also responsible for difference in results.

Among bacteria some strains or races are more resistant to heat than others. These differences, which correspond to similar known variations in all animal and vegetable species, must be taken into account.

Evaporation takes place so rapidly from exposed fluids that the surface layer may remain cooler than the body of the liquid. This is especially a matter of concern with milk, which sometimes forms a scum above 60° C., owing to rapid evaporation of the surface layer. This scum consists of coagulated albumins in which is enmeshed much fat. The bacteria entangled in this surface pellicle may escape the heat indicated by the thermometer in the deeper layers.

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[a] This article is a brief summary of Hygienic Laboratory Bulletin No. 42, entitled "The thermal death points of pathogenic micro-organisms in milk." For all the details of the work the reader is referred to that bulletin.

As a rule, bacteria are attenuated and lose their power to infect before they lose their ability to vegetate upon artificial culture media. It is therefore safe to assume that a micro-organism that will not grow in artificial media under favorable conditions is "dead." The tubercle bacillus is an exception to this rule, for reasons given further on.

The methods used in the tests recorded below were planned to imitate the actual conditions of pasteurization, so far as practicable, in laboratory experiments.

The test tubes in which the infected milk was heated were open to the air, and scum formation was disregarded in all instances, my object being to determine the thermal death point against natural difficulties, so that the results might be applied with confidence to practical pasteurization.

BACILLUS TUBERCULOSIS.

Certain special difficulties are met with in determining the thermal death point of the tubercle bacillus. This organism does not grow readily upon artificial media. The few experiments made to determine its thermal death point by cultural methods have no significance, because its vegetability upon artificial media does not correspond to its power of growing in the animal organism. It is therefore necessary to inoculate animals in order to determine whether or not the tubercle bacillus is alive and virulent. Here again we meet with complications. Dead tubercle bacilli have a certain amount of pathogenic power and produce lesions, including tubercle formation, abscesses, and coagulation necrosis. However, while we lack a criterion to determine with precision the exact point when the tubercle bacillus dies, we are able by means of animal inoculations to determine just when the tubercle bacillus is so enfeebled that it is no longer able to infect. This, after all, is the important practical point.

In my own experiments, in order to avoid the confusion resulting from the effects produced by dead tubercle bacilli, doubtful lesions were carried over into another animal.

From these experiments it is evident that the tubercle bacillus in milk loses its infective properties for guinea pigs when heated to 60° C. and maintained at that temperature for twenty minutes or to 65° C. for a much shorter time.

It should be remembered that the milk in these tests was very heavily infected with virulent cultures, indicated by the prompt deaths of the control animals. Milk would practically never contain such an enormous amount of infection under natural conditions. It is justifiable to assume that if 60° C. for twenty minutes is sufficient to destroy the infectiveness of such milk when injected into the peritoneal cavity of a guinea pig, any ordinary market milk after such treatment would be safe for human use by the mouth so far as tubercle bacilli are concerned.

It is difficult, if not impossible, to briefly summarize the work of others upon the thermal death point of the tubercle bacillus in milk. The following table necesarily leaves out many factors:

Table showing the thermal death point of the tubercle bacillus as found by various investigators.

Investigator.	Killed at—	Not killed at—
Martin, 1882		80°
May, 1883	By cooking	
Sormani, 1884	Boiling, 5 minutes	60°, for 10 minutes.
Schill and Fisher, 1884		100°.
Voelsch, 1887		100°, boiling twice.
Yersin, 1888	60°, 10 minutes (−spores) 60°, 10 minutes (+spores)	
Bitter, 1890	68°, 20 minutes	
Bang, 1891	70°, 5 minutes (enfeebles) 60°, 5 minutes (sometimes enfeebles). 80° (sometimes kills) 85° (always kills)	
Bonhoff, 1892	60°, 20 minutes	50°, 60 minutes.
Gancher and Ledeux-Lebard, 1892	60°, 5 minutes (attenuates) 70°, 1 minute (kills)	
Forster, 1892	60°, 6 hours 95°, momentary	55°, 3 hours. 60°, 45 minutes. 80°, momentary.
De Man, 1893	55°, 4 hours 60°, 1 hour	60°, 45 minutes.
Schroeder, 1894	60°, 15 minutes	
Woodhead, 1895	50°, 15 hours 60°, 8 hours 60°, 45 minutes 70°, 45 minutes 70°, 2½ minutes	90° (results contradictory).
Marshall, 1899	68°, 20 minutes	60°, 10 minutes.
Th. Smith, 1899	60°, 15 to 20 minutes	
Morgenroth, 1900	55°, 3 hours	70°, 10 minutes. 100°, momentary.
Kobrak, 1900	50°, 4 hours	
Beck, 1900	100°, 3 hours	100°. 80°, 30 minutes.
Galtier, 1900		85°, 6 minutes.
Russell and Hastings, 1900	60°, 20 minutes	
Herr, 1901	65°, 15 minutes	80°, 5 seconds.
Hesse, 1901	60°, 20 minutes	
Levy and Bruns, 1901	65°, 15 minutes	
Berthel and Stenstrom, 1901		70°, 15 minutes.
Bang, 1902		60°, 15 minutes.
Tjaden, 1903	85°, 1 to 2 minutes	
Rullmann, 1903	65°, 30 minutes	60°, 20 minutes.
Barthel and Stenström, 1904	80°, 1 minute (uncoagulated)	80°, 1 minute (coagulated)
Russell and Hastings, 1904	71°, 1 minute	
Zelenski, 1906		76°, 20 minutes.
Rosenau, 1907	60°, 20 minutes	

The above tabular statement shows that my results agree with the work of Yersin, Bonhoff, Schroeder, Th. Smith, Russell and Hastings, and Hesse in that 60° for twenty minutes is sufficient to kill the tubercle bacillus.

The lesions produced by a large mass of dead tubercle bacilli may be distinguished by their extent rather than by their character. In doubtful cases secondary inoculation is the only trustworthy method of determining whether the bacilli are alive or dead. The tuberculin test does not differentiate between the live and dead tubercles. Three guinea pigs out of eight having lesions produced by dead tubercle bacilli (killed at 100° C.) died as the result of the subcutaneous inoculation of 2 c. c. tuberculin (O. T.).

CONCLUSIONS.

The evidence is plain that milk heated to 60° C. and maintained at that temperature for two minutes will kill the typhoid bacillus. The great majority of these organisms are killed by the time the temperature reaches 59° C., and few survive to 60° C.

The diphtheria bacillus succumbs at comparatively low temperatures. Oftentimes it fails to grow after heating to 55° C. Some occasionally survive until the milk reaches 60° C.

The cholera vibrio is similar to the diphtheria bacillus so far as its thermal death point is concerned. It is usually destroyed when the milk reaches 55° C.; only once did it survive to 60° C. under the conditions of the experiments.

The dysentery bacillus is somewhat more resistant to heat than the typhoid bacillus. It sometimes withstands heating at 60° C. for five minutes. All are killed at 60° C. for ten minutes. However, the great majority of these micro-organisms are killed by the time the milk reaches 60° C.

So far as can be judged from the meager evidence at hand, 60° C. for twenty minutes is more than sufficient to destroy the infective principle of Malta fever in milk. The *M. melitensis* is not destroyed at 55° C. for a short time; the great majority of these organisms die at 58°, and at 60° all are killed.

Milk heated at 60° C. and maintained at that temperature for twenty minutes may therefore be considered safe so far as conveying infection with the micro-organisms tested is concerned.

23. INFANT FEEDING.

INFANT FEEDING.

By Joseph W. Schereschewsky,

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PART I.—INFANT MORTALITY IN RELATION TO INFANT FEEDING.

Owing to the long duration of the period of infancy in human beings, as compared to that of the lower animals in general, it is obvious that the opportunity of environment to react upon our development is enormously increased over that afforded in the case of other living beings.

The effect of prenatal influences upon our ultimate development receives no further accretions from the moment of our birth, and, apart from those congenital defects and states of debility, whose influence upon life are manifest from the outset, our subsequent growth and development are almost exclusively controlled by our immediate surroundings.

More than any other component factor of its environment, food, the form and the methods of its administration, are capable of influencing the future development and determining the fate of the newborn child.

If this statement be true, we should expect to find that an investigation of the mortality rates of infants would furnish some relevant facts in regard to this question.

Unfortunately, even at the present time infant mortality and the degree to which such mortality is influenced by improper methods of feeding is not a subject of general knowledge. True, it is known as a matter of casual information that the rate of mortality among the newborn is relatively high, yet few who have not paid attention to the matter realize, as Bergeron so graphically puts it, that the chances of a newborn child surviving a week are less than those of a man of 90; of living a year, less than those of a man of fourscore.

Information as to the infantile death rate in this country is difficult to derive, owing to the small number of States within our registration area and the poverty in detail of their statistical returns. The writer is, however, much indebted to Harrington, who has made an extensive study of this question in a recent article from which many of the following figures and facts are taken. The perusal of his article is recommended to those who desire a more extended treatment of this phase of the subject.[a] Reference to the report of the Bureau of Census for 1900 shows that the general infantile mortality rate per thousand in the States which constituted our registration area at that time is as follows:

TABLE 1.

District of Columbia	274.5	New York	159.8
Rhode Island	197.9	Connecticut	156.8
Massachusetts	177.5	Maine	144.1
New Hampshire	172.0	Vermont	122.1
New Jersey	167.4	Michigan	121.1

These figures show the wide variations to which the infantile mortality rate is subject in different parts of this country. The lowest mortality rate, 121.1 (Michigan), is less than half the highest (Washington, D. C.).

On comparing them with foreign countries, however, they do not strike us as extraordinarily high. The death rate of Washington, D. C., is similar to that of Russia, while the rate of the lowest (Michigan) corresponds to that of Scotland, but exceeds that of three other European countries, viz, Ireland (average of twenty years, 1874 to 1893, 96.6), Norway (1902, 75.08), and Sweden (1902, 87).

As is to be expected, our cities show a higher infant mortality rate than our States. The census report of 1900 gives 106 towns and cities which have an infant mortality of 175 or over. The maximum infant death rate was shown by Charleston, S. C., with a rate of 419.5, while Los Angeles, Cal., with a rate of 175, occupied the last place.

The following cities showed a death rate of over 300 per 1,000 births:

TABLE 2.

Charleston, S. C	419.5	Atlanta, Ga	306.0
Savannah, Ga	387.5	Fall River, Mass	304.7
Mobile, Ala	344.5	Lynchburg, Va	301.7
Key West, Fla	311.8	Richmond, Va	300.1
Biddeford, Me	311.6		

The following large representative cities had an infantile mortality rate as follows:

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[a] Harrington, Am. Jour. Med. Sci., Vol. CXXXII, pp. 811–835.

TABLE 3.

Washington, D. C	274.5	Brooklyn, N. Y	197.2
Baltimore, Md	235.1	Boston, Mass	194.1
New Orleans, La	229.2	Borough of Manhattan	190.9
Philadelphia, Pa	197.2	New York, N. Y	189.4

The poverty of our vital statistics does not, however, permit us to analyze these figures as to the incidence of various infantile conditions and diseases causing death. In order to interpret their significance, we must resort to the statistics of foreign countries, most of which have complete and excellent systems for the registration of death returns.

Inasmuch as in civilized countries similarly situated with respect to latitude the circumstances affecting mortality are approximately the same, it may be postulated that conditions shown to exist abroad are duplicated here.

Owing chiefly to her falling birth rate, the state of infant mortality in France has for some years been a subject of acute interest both to her Government and to her medical profession. We find that the average infantile mortality rate of France has been 167 for the twenty-year period of 1874–1893. In 1903 this rate had fallen to 137, and contrary to what is usually the case was lower than this in some of her largest cities, such as Lyon (110), Bordeaux (102), and Paris (101).

In January, 1901, Balestre and Gileta de St. Joseph presented a memoir to the Académie de Médecine dealing with the infant mortality of France from the years 1892 to 1897, with special reference to the various causes of death constituting the total infant mortality rate.[a]

Their analysis showed that in every 1,000 infant deaths under 1 year of age no less than 385 were due to gastro-intestinal disease, 171 were due to congenital states of debility, 147 to disease of the respiratory organs, 50 to acute contagious disease, 25 to tuberculosis, and 222 to all other causes. This was the average for the whole country. In certain cities the death rate from gastro-intestinal disease was enormously increased, being 700 per thousand infant deaths in the city of Troyes in 1892.

In 1905 Ausset[b] published a report on the infant mortality of the Département du Nord practically confirming the figures of Balestre and Gileta de St. Joseph.

[a] Rept. by M. Perret, Revue d'Hygiéne et de Méd. Infantiles, 1905, IV, 160.

[b] E. Ausset, Revue d'Hygiéne et de Med. Infantiles, 1905, II, 433. (Cited by Harrington.)

The following table gives the number of deaths from gastro-intestinal diseases per thousand deaths in infants of less than 1 year of age in certain parts of the Département du Nord:

TABLE 4. *Deaths from gastro-intestinal disease per 1,000 infant deaths, Département du Nord, France.*

District of Dunkerque	445. 08	District of Lille—Continued.	
Canton Bergues	410. 61	3 cantons of Tourcoign	457. 77
Canton Bourbourg	485. 38	8 cantons of Lille	427. 83
Canton Wormhoudt	602. 86	District of Hasebrouck	395. 20
Canton Gravelines	404. 40	Canton Merville	363. 60
2 cantons of Dunkerque	465. 00	Canton Steenvoorde	394. 44
District of Lille	401. 26	Canton Cassel	454. 66
Canton Armentières	416. 66	3 cantons of Bailleul	431. 11
Canton Quesnoy-sur-Lille	420. 28	District of Valenciennes	313. 92
Canton Seclin	383. 37	Canton Denain	342. 10
Canton Launay	398. 87	Canton Condé	337. 07
Canton Rontaix	464. 33	Canton Bouchain	392. 40

In Germany, which has the second highest infantile mortality rate in Europe, being surpassed "in this bad eminence" only by Russia (which in some districts has an infantile mortality rate surpassing 500, and for the whole country one of 270), we find that the infantile mortality rate for the quinquennium of 1901–1905 is as follows:

TABLE 5. *Average infant mortality under 1 year per 1,000 births in Germany, 1901–1905.*

1901	207	1904	196
1902	183	1905	205
1903	204		

In 1904 the average rate for 323 German cities and towns having a population of 15,000 or over was 202; in 1905 it was 204.

In the latter year the average rate of 42 German cities each with a population exceeding 100,000 was 202, and for the twelve months ending June 30, 1906, was 198. The returns of that year from these 42 German cities show further that of 67,637 infant deaths no less than 28,423, or 44.03 per cent, were due to diarrheal disease.

The excellence of the German system of registration of vital statistics permits us to examine the local incidence of deaths from gastro-intestinal disorders.

I have taken the liberty of combining two of the tables in Harrington's[a] admirable article into the following, which shows the birth rate, the diarrheal death rate, and, finally, the percentage the diarrheal death rate constitutes of the total infantile mortality in the 42 German cities listed in the table for the twelve months ending June 30, 1906:

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[a] Harrington, Am. Jour. Med. Sci., Vol. CXXXII, pp. 811–35.

TABLE 6.—(After Harrington.)

Name of city.	Births, July 1, 1905, to June 30, 1906.	Deaths under 1 year of age.	Death rate per 1,000 births.	Diarrheal death rate.	Per cent of deaths due to diarrhea.
Aachen	4,300	839	195	78.60	40.23
Altona	4,205	710	169	38.37	22.64
Barmen	4,597	605	132	47.64	36.20
Berlin	49,708	9,933	200	87.99	44.03
Borkum	5,060	818	162	50.20	31.05
Bremen	6,420	1,116	174	73.92	42.52
Breslau	14,366	3,511	244	98.62	40.56
Brunswick	3,397	668	197	81.22	41.32
Cassel	3,202	407	127	31.85	25.06
Charlottenburg	5,189	803	155	54.35	35.12
Chemnitz	8,314	2,253	271	135.67	50.07
Cologne	15,403	3,266	212	93.60	44.06
Crefeld	2,752	390	142	53.42	37.69
Dantzig	5,288	1,286	243	109.87	45.18
Dortmund	7,385	1,366	185	61.75	33.38
Dresden	14,297	2,735	191	83.23	43.51
Duisberg	7,524	1,137	151	97.38	44.59
Dusseldorf	8,868	1,667	188	83.43	43.85
Elberfeld	4,966	739	149	50.97	34.23
Erfurt	2,966	597	202	42.90	21.27
Essen	9,494	1,498	158	66.67	42.26
Franfort-on-the-Main	9,335	1,446	155	53.13	34.30
Gelsenkirchen	7,451	1,169	157	58.11	37.04
Halle	4,985	1,175	236	124.77	52.91
Hamburg	20,471	3,538	173	70.98	41.07
Hanover	5,908	949	161	37.41	23.28
Karlsruhe	3,052	565	185	86.17	46.55
Kiel	5,083	903	178	72.99	41.08
Koenigsberg	6,671	1,556	[illegible]	113.47	[illegible]
Leipzig	14,734	3,273	222	121.49	[illegible]
Magdeburg	6,304	1,472	[illegible]	100.23	[illegible]
Mannheim	5,170	1,053	204	86.46	42.45
Munich	15,787	3,432	217	95.33	44.11
Nuremberg	10,290	2,547	248	113.80	[illegible]
Plauen	3,745	779	208	35.25	16.95
Posen	5,123	1,272	248	95.52	38.47
Rixdorf	5,547	1,212	218	103.66	47.44
Schoeneberg	[illegible]	480	140	49.38	[illegible]
Stettin	7,089	1,847	261	112.28	[illegible]
Strassburg	4,794	915	190	92.82	48.74
Stuttgart	6,176	1,270	206	74.80	[illegible]
Wiesbaden	2,489	[illegible]	162	40.52	[illegible]
	[illegible]	67,637	198	[illegible]	[illegible]

An examination of this table shows that the highest infantile death rate recorded for these 42 German cities is that of Chemnitz, with a total infantile death rate of 271 and a death rate from diarrheal diseases of 135.67. As Harrington points out, this latter is greater than the total infantile death rate of the city of Barmen (132), and is greater than the total infantile death rate of England and Wales

(125 in 1903), Scotland (1900–1901, 128, highest in 50 years), Norway (105, average 20 years 1874–1893), Sweden (87, 1902), Ireland (96.6, average 20 years 1874–1893), and is almost as high as the total death rate of France (137 in 1903). The average death rate from diarrhea in all these 42 cities was 80.34—figures of great significance when we come to investigate the seasonal distribution and the cause of the prevalence of gastro-enteritis in the neonate.

It is worthy of note that the census report of 1900 shows that in our country there are no less than 15 cities having a total death rate greater than that of Chemnitz, viz:

TABLE 7.

Charleston, S. C.	419.5	Richmond, Va	300.7
Savannah, Ga	387.5	Laconia, N. H	294.6
Mobile, Ala	344.5	Shreveport, La	293.5
Key West, Fla	311.8	Jacksonville, Fla	287.6
Biddeford, Me	311.6	Norfolk, Va	284.6
Atlanta, Ga	306.0	Lowell, Mass	275.5
Fall River, Mass	304.7	Washington, D. C.	274.5
Lynchburg, Va	301.7		

The figures in connection with the German cities concern places of a population in excess of 100,000, while the cities returning rates in this country are, many of them, considerably smaller than this. As Harrington points out, our methods of registration are so incomplete that full returns would probably indicate a condition worse than now is manifest, and that we have every reason to suppose, in view of the extreme heat of our summers, that the diarrheal death rate in this country forms at least as great a proportion of the total mortality as it does abroad.

SEASONAL FLUCTUATION.

Nor does the infantile death rate maintain itself constantly throughout the year at the same general level. On the contrary, it is well known that it is subject to enormous fluctuations, being extremely high during the months of July, August, and September, followed by a sharp decline in the autumn. This accession to the infant death rate is due to the great number of deaths from diarrhea alone in those months, as the rate of mortality due to other infantile diseases remains pretty constant throughout the year.

For example, at Leipzig, whose percentage (54.9) of deaths from diarrhea is higher than that of any other city in Germany, a comparison of the birth rate, the infantile death rate, and the diarrheal death rate by months shows that in August, with an infant death rate of 570 to 1,000 births, 430 of these, or 75.6 per cent, were due to gastro-enteritis, whereas in February the total infantile mortality had sunk to 131, of which diarrheal diseases constituted a proportion of only 37 to a thousand, a decrease of 1,100 per cent.

In England the report of the registrar-general's office, compiled from the weekly returns of births and deaths from 76 of the largest cities of England and Wales in July, August, and September, 1906, shows that the total births for these months were 110,209, the total deaths under 1 year 23,058, of which no less than 14,306, or over 50 per cent, were due to diarrhea.

It is manifest from the foregoing that gastro-intestinal disease, causing as it does one-third to one-half of all infant deaths under 1 year of age, is the largest single factor determining infant mortality. Further investigation demonstrates the significant fact that 75 to 85 per cent of all infants who die of diarrhea are artifically fed. Thus Planchon, in investigating the relation feeding methods had to gastro-enteritis in Paris, shows[a] that, while the diarrheal death rate in breast-fed infants varies from a minimum of 2 per thousand in winter to a maximum of but 20 per thousand during the hot months, the diarrheal deaths of the artificially fed fluctuate from a minimum of 12 per thousand in winter to a maximum of 158 per thousand in the summer.

In Paris during the four summer months of 1897, 2,840 infants under 1 year died. Of these 1,470, or 51.7 per cent, died of diarrhea. Of these 1,470 who died of diarrhea, only 139 were breast fed, and 1,331, or over 90 per cent, were artificially fed.

The following table from Harrington (loc. cit.) illustrates admirably this point at Berlin. The figures given cover the quinquennium of 1900-1904 and relate to the incidence of deaths among the bottle fed and the breast fed when the method of feeding could be determined.

TABLE 7.

Year.	Number of deaths among infants under 1 year of age whose mode of feeding was known.	Number of deaths among breast fed.	Percentage of deaths of breast fed infants.	Percentage of deaths among others.
1900	9,558	895	9.36	90.60
1901	9,378	856	9.17	90.80
1902	7,027	768	10.17	89.10
1903	7,680	723	9.41	90.59
1904	7,780	714	9.18	90.82

Again, Helle[b] in analyzing the infantile death rate of the city of Graz shows that out of 170 deaths from intestinal disease in the fis-

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[a] Planchon: Prevalence of Diarrhea in the Artificially Fed. Obstetrique, January, 1900.

[b] K. Helle: Archiv f. Hygiene, 1905, VI, 18.

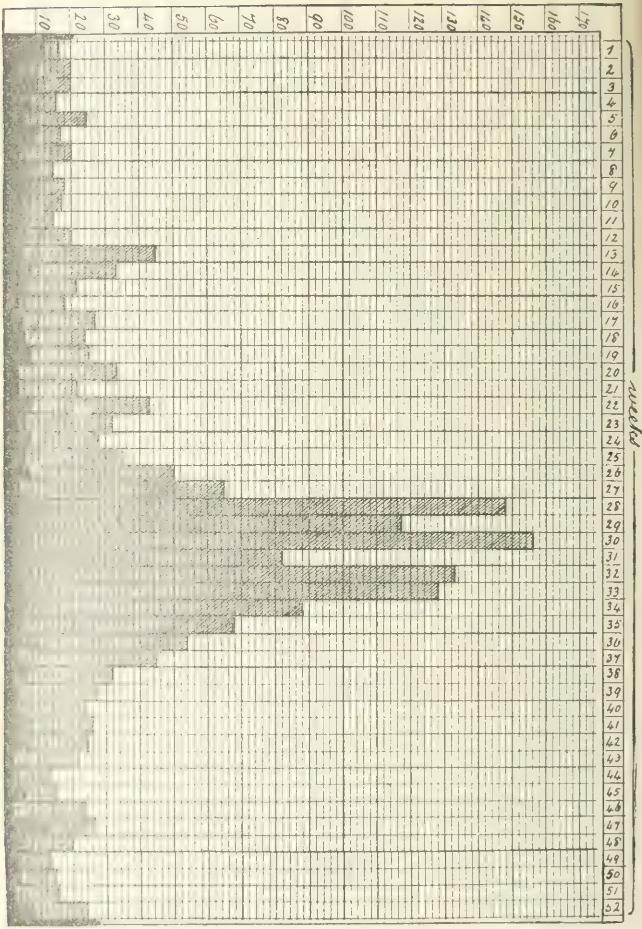

Fig. [illegible]. — Chart showing the relative mortality from gastro-intestinal disease in breast fed and bottle fed infants, 0–1 year of age, in Paris, by weeks and throughout the year.

Breast fed infants ▨.
Bottle fed infants ☐.

cal year of 1903–4 but 4 of these were breast fed, 48 were partly breast fed, 117 were bottle fed, and in one case the method of feeding was unknown.

Reference to Table 6 shows that the German city of Barmen, with an infant mortality rate of 132 (1906), enjoys the lowest rate of any city in Germany, and for a number of years has made a favorable showing in this respect. Kriege and Sentemann[a] attribute this fortunate circumstance to the general prevalence of breast feeding in that city, 63 per cent of all infants being nursed by their mothers, 15 per cent being partly breast fed, and only 22 per cent being bottle fed.

Further space can not be devoted to the multiplication of figures showing the relative immunity of the breast-fed child to death from diseases of the digestive tube. Nothing, however, can more graphically illustrate this point than the accompanying chart from Budin which is here reproduced (p. 696).

PART II.—THE INFANT'S DIETARY.

In common with adults, the infant requires five elements of food for its sustenance, to wit: Proteid, carbohydrate, fat, mineral salts, and water. Owing, however, to the undeveloped state of its organs of assimilation it can not avail itself of any wide dietary range. By reason of its rapid growth and more active metabolism it requires food of special form and with the nutritive ingredients in special proportions to each other. Milks are the only class of food which fulfill these conditions, being, as they are, an animal product, designed by nature only to that end.

As this paper deals merely with the dietary of infants less than 1 year of age, woman's milk and its only feasible substitute, cow's milk, will alone be considered.

WOMAN'S MILK.

Woman's milk is the secretion of the human mammary gland. Under normal conditions of lactation it is in no sense a transudation from the blood and lymphatics, but is a true secretion elaborated by glandular tissue. True milk is not present in the mammary glands until two to four days after parturition, and occasionally not until the fifth day.

Colostrum.—The secretion present in the mamma for the first few days after delivery differs materially from normal milk and is known as "colostrum." It is a fluid of a deep yellow tint, chiefly due to bodies it contains known as "colostrum corpuscles." It is not so sweet as milk, is strongly alkaline in reaction, of a specific gravity of 1.030 to 1.040, and is rich in salts and proteids. These proteids are of a nature similar to the proteids of the blood as they are coagulated by heat. Colostrum contains less sugar and fat than milk, and microscopically its fat globules vary in size and are interspersed with numerous bodies four or five times their size, known as "colostrum corpuscles."

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[a] Allg. Centralblatt f. Gesundheitspflege, 1900, XXV, 25.

Composition of colostrum.—According to Pfeiffer's analysis, the composition of colostrum is as follows:

	Per cent.
Proteid	5.71
Fat	2.04
Sugar	3.74
Salts	0.25
Water	88.23
	100.00

Caloric value per kilogram, 577.17 calories.

The colostrum corpuscles are very abundant during the first few days, but under normal conditions disappear after the tenth or twelfth day.

Function of colostrum.—The exact rôle of the colostrum is not as yet fully understood. We may infer from the nature of its composition and its proteids that it furnishes to the newborn child during its adjustment to its novel surroundings the full expansion of the lungs and the awakening of the digestive processes, nourishment of a character similar to that it received from the placenta as a fetus. That it serves a purpose is proven by its being the first secretion not only of the human breast but of that of all mammals.

Physical characteristics of woman's milk.—With the establishment of lactation the breast secretes a fluid of the following physical characteristics: It is of a bluish color and marked sweetish taste. Under normal conditions, with the exception of some skin cocci, it is practically sterile. These are most abundant in the "foremilk." Its specific gravity varies from 1.026 to 1.036 (average, 1.032 at 21° C.). Its reaction is either amphoteric or slightly alkaline when fresh. Dilute acetic acid merely produces a light flocculent precipitate, and its proteids are not appreciably coagulated by the action of rennet.

Composition.—The exact average composition of breast milk is difficult to determine, as it is subject to rather wide variations between normal limits and at different stages during the act of being secreted. Thus the "foremilk" is relatively thin, the middle portion richer, and the "strippings" richest of all in fat content. Owing, moreover, to faulty methods, previous analyses of woman's milk have been erroneous. Even now its exact composition, beyond the relative proportions of its constituents, is imperfectly understood.

According to the most recent analyses of Pfeiffer, Koenig, Leeds, Harrington, Adriance, and others the average composition of human milk is as follows:

Composition of woman's milk.

	Percentage.	Common normal variations.	
		Per cent.	
Fat	4.00	3.00–	5.00
Sugar	7.00	6.00–	7.00
Proteids	1.50	1.00–	2.25
Salts	.20	.18–	.25
Water	87.30	89.82–	85.50
	100.00	100.00	100.00

An average caloric value per kilogram, 710.5 calories; common normal variations of caloric value per kilogram, 550 calories to 844.25 calories.

Former analyses have for the most part assigned to it a higher amount of proteid and a lesser amount of sugar than this. The composition of milk is pretty nearly constant throughout lactation, except during the first month and toward the close. At the commencement of lactation the proteids and salts are high, and near its end the proteids have a tendency to diminish (Adriance).

Proteids.—Our knowledge of the proteids of woman's milk is still incomplete. The most important proteid substances, however, are casein and lactalbumen. Some investigators mention a third, lactoglobulin. The casein is in chemical combination as calcium casein, and owing to its relative proportions to the other proteids is only slightly precipitated by dilute acids and not appreciably coagulated by rennet.

The lactalbumen is believed to be similar to serumglobulin.

The proportions of lactalbumen to casein have not been definitely agreed upon, but it exists in far greater proportions relative to the casein than in cow's milk. According to Koenig, the relative proportions are as 5 to 1. The total amount of proteids varies normally from 1 to 2 per cent and abnormally from 0.07 to 1.5 per cent. They are highest during the first few days of lactation; after the first few weeks they vary but little until toward its end, when they experience a decided decrease.

Fat. Fat is present in woman's milk in the form of minute globules, and in perfect emulsion by virtue of the albuminous fluid in which they are suspended. It exists mainly in the form of the neutral fats, olein, palmitin, and stearin, and but small quantities of the fatty acids are present. Forty-three analyses by Leeds show variations in the fat content of woman's milk of between 2.11 and 6.89 per cent, with an average of 4 per cent. The percentage of fat present in woman's milk is but little affected by the period of lactation.

Sugar.—Sugar is the most constant of the ingredients of human milk in its percentage. It is present as lactose in complete solution in the proportions of from 6 to 7 per cent. Its quantity is least in the first week. After the first month its variations are very light.

Salts. Only one-fourth as much inorganic salts is present in woman's milk as in cow's milk, and, with the exception of the calcium in combination with the casein, are all in solution. They are present in the proportion of 20 per cent.

CLINICAL EXAMINATION OF WOMAN'S MILK.

It is often of importance to recognize the occurrence of quantitative and qualitative departures from the normal composition of woman's milk occurring during lactation, and their nature, as upon them are dependent many nutritional disturbances of the nursing child.

The most common abnormalities to be recognized are (*a*) disturbances in the quantity and (*b*) disturbances in the quality of the lacteal secretion.

According to the researches of Haehner, Feer, Huebner, Laure, Ahlfeld, and others, the average daily quantity of milk drawn by infants of different ages is as follows:

	Ounces.
At end of first week, 300 to 500 grams	10 to 16
During second week, 400 to 550 grams	13 to 18
During third week, 430 to 720 grams	14 to 24
During fourth week, 500 to 800 grams	16 to 26
From fifth to thirteenth week, 600 to 1,030 grams	20 to 34
From fourth to sixth month, 720 to 1,150 grams	24 to 38
From sixth to ninth month, 900 to 1,220 grams	30 to 40

The average daily amount of milk per kilo of body weight drawn by the child was found to be as follows:

	Ounces.
During first three months, 150 cubic centimeters	5
During second three months, 140 cubic centimeters	4⅔
During third and fourth three months, 120 cubic centimeters	4

It was also found that the total daily amount drawn corresponds very nearly to the following figures in proportion to the body weight of the child:

First three weeks, one fifth of body weight.
First month to end of sixth month, one-sixth to one-seventh of body weight.
Last half of first year, one-eighth of body weight.

The daily quantity of the milk drawn from the breast by the child is best determined by weighing the child before and immediately after each feeding during the entire twenty-four hours for several days. An accurate set of scales, sensitive to 15 grams (½ ounce) should be used. By computing the sum of the weights of the separate feedings for each day and striking an average for the daily amounts during the period of observation, the average amount of the daily consumption of milk can then be determined. As children vary in age, weight, and nutritive needs, the figures obtained will only be of value when compared to the body weight and age of the child that received them, as is subsequently to be discussed.

Reaction.—This may be tested by litmus paper and should be alkaline or amphoteric, never acid.

Specific gravity.—This may be determined with the aid of any small hydrometer, such as a urinometer with a scale registering from 1,010 to 1,040. The specific gravity is lowered by fat, but increased by the other solids.

Microscopical examination.—Besides the fat globules, the microscope may reveal the presence of colostrum corpuscles, blood, pus, epithelial cells, bacteria, and granular detritus. The presence of colostrum corpuscles is abnormal after the twelfth day of lactation. Blood and pus are always abnormal. The presence of blood and pus in the milk require the suspension of lactation until they disappear.

Determination of fat.—The simplest method of determining the fat of woman's milk is by Holt's cream gauge. This is a graduated tube on a foot, with a glass stopper. The tube is filled with freshly drawn milk to the zero mark at the top of the scale and the whole allowed to stand at room temperature for twenty-four hours. The percentage of cream according to the scale is then read off. The ratio of the cream to the fat content is as 5:3; e. g., 5 per cent of cream equals 3 per cent of fat, etc.

While not very accurate, this method suffices for clinical purposes. Results approximating the accuracy of a chemical analysis may be obtained by the Babcock test or by Lewis's modification of the Leffman and Beam test for cow's milk (Holt). This is a centrifugal test for which special tubes are required, which, however, may be used in the ordinary centrifuge for urine.

Sugar.—The percentage of sugar in human milk is subject to very little variation, and may be regarded as constant for clinical purposes.

Proteids.—The determination of the proteids in woman's milk is an elaborate process requiring the resources of a well-equipped chemical laboratory.

We may, however (according to Holt), gain an approximate idea of their percentage by considering the sugar and salts of milk as constants not affecting its specific gravity, and estimating the proteids from our knowledge of the fat content of the specimen. Now, the specific gravity will vary directly with the proteids and inversely to the fat, viz, high proteids, high specific gravity; high fat, low specific gravity. The following table shows the application of this principle:

Variations in the composition of woman's milk as deduced by observation of the specific gravity and the fat content (Holt).

	Specific gravity, 70° F.	Cream, 24 hours.	Proteids, estimated.
Average	1,031	7 per cent	1.50 per cent.
Normal variations	1,028–1,032	8–12 per cent	Normal (rich milk).
Normal variations	1,031	5–6 per cent	Normal (fair milk).
Abnormal variations	Low (below 1,028)	High (above 10 per cent)	Normal or slightly below.
Abnormal variations	Low (below 1,028)	Low (below 5 per cent)	Low (very poor milk).
Abnormal variations	High (above 1,032)	High	Very high (very rich milk).
Abnormal variations	High (above 1,032)	Low	Normal or nearly so.

As the milk drawn from the breast during the first part of nursing is richer in proteids and much poorer in fats and the last portion rather poorer in proteids and rich in fats, the entire amount of milk present in the breast should be drawn off for the purpose of this estimation.

COW'S MILK.

Cow's milk is the only food supply, apart from mother's milk, available in this country, from a practical standpoint, for the nourishment of infants under 1 year of age. It forms besides a large part of the dietary of older children and of many adults. It is consequently of the utmost importance, in view of its perishability, that it should only be used as a food under conditions which will insure its wholesomeness.

We have already considered the enormous loss of life occurring among the artificially fed infants, of which the larger part is undoubtedly due to bad milk and its improper use as an article of diet.

Stated as a general proposition, the following conditions should be fulfilled in milk that is to be used as a basis for the nourishment of young infants: First, it should be clean; second, it should be fresh; third, it should be whole (i. e., not falsified by additions or subtractions of its component parts or by the addition of preservatives); fourth, it should be free from pathogenic organisms and toxic products; and fifth, it should be kept cold.

Importance of clean milk. By clean milk we understand a milk which has been collected under such hygienic conditions from healthy animals and handled under such precautions as to insure its reaching the consumer without containing any visible particles of extraneous matter as well as any excessive number of bacteria.

Unfortunately, whenever the milk supply of a community has been investigated, either under public or private auspices, the conditions found to prevail in the production and handling of milk have always been disappointing, if not, as in many instances, revolting to the last degree. The insanitary surroundings and general condition of filth prevailing at some dairy farms is at times indescribable, and the examination of milk produced under these conditions reveals not only a bacterial flora, but a degree of contamination with gross particles of extraneous matter such as to suggest utter carelessness or ignorance on the part of the producer.

Milk when produced under such circumstances not only contains a plentiful enrichment of dust, dirt, dung, cow hairs, flies, and other foreign bodies, but also a bounteous inoculation of bacteria of all forms, such as may render it from the very outset unfit for human consumption.

Significance of a large bacterial content in milk.—While many of the numerous varieties of bacteria encountered in milk are of a harmless character, their presence in large numbers is always evidence of either milk carelessly handled or milk improperly cooled and kept. The presence of gross contamination with the foreign matters previously enumerated insures the planting of the bacteria of putrefaction and decomposition. Such milk, without suffering any material change in its taste or physical appearance, may contain the poisons of bacterial activity to a dangerous extent. No universal standard has as yet been settled upon as to what constitutes an excessive degree of bacterial contamination of milk. In general it has been agreed that for milk sold from cans, anything less than 100,000 to the cubic centimeter is good; for milk sold in bottles, anything under 10,000 must be considered especially good. Yet it is possible by the exercise of especial care to produce a milk which the year round when delivered to the consumer will have an average bacterial content of less than 5,000 to the cubic centimeter. Milk from high grade dairies, when sold in bottles, usually averages from 10,000 to 100,000 bacteria, while milk sold from cans may range anywhere from 100,000 to 40,000,000, especially in hot weather.

Fresh milk.—By fresh milk, we understand milk less than twenty-four hours old when delivered. Under the ordinary conditions prevailing in the handling of milk it will have undergone such fermentative changes as to render it unfit for the use of young children after the expiration of this period. The special conditions of care in the production of milk which render it safe after a longer time than this are unfortunately very far from prevalent.

Infected milk.—Infected milk is milk contaminated with pathogenic germs. Very many instances have been and are still being adduced of epidemics of the zymotic diseases, such as typhoid fever, scarlet fever, diphtheria, and the like, which have been directly traced to milk contaminated with their specific bacilli. Such epidemics originate either in the water supply of the dairy farm or from sickness among the personnel engaged in handling it. Pathological conditions affecting the cow are also contributive to the infection of milk. Thus tuberculous disease of the udder is a fruitful source of the presence of the bacillus of tuberculosis, and garget, an inflammation of the bovine mammary gland, is a very common cause of the presence of pus and streptococci. In short, unless conditions affecting the water supply, the dairy farm, the health of the cattle, and the incidence of disease among the employees engaged in handling it from the cow to the consumer are subject to efficient prophylaxis, so long will conditions favorable to the infection of milk obtain.

Falsification of milk.—It is obvious that milk should be what it purports to be, i. e., whole milk. It should, therefore, not be robbed of its content of butter fat by skimming, nor should its color be improved by artificial means. It is needless to say that no preservatives should be added to it, as is often done by the unscrupulous.

Cold milk.—The prompt cooling of milk, after it is drawn, to a temperature of 7.7° C. (45° F.) and its maintenance at that temperature until the time of consumption is one of the most efficient means at our command for restraining the growth of its bacterial content and preserving its wholesomeness as a food. Milk is an excellent culture medium for bacteria. Although by appropriate precautions the number of germs present can be very materially reduced, there are no methods at present commercially possible by which the natural sterility of mother's milk as drawn by the babe can be imitated. It is entirely within our means, however, to restrict the growth of bacteria in milk.

The rapid cooling of cow's milk after it has been drawn to a temperature of 7.7° C. (45° F.) and the maintenance of that temperature until the time of consumption not only prevents the multiplication of its original bacterial content, but actually tends to diminish their number. In the winter, the low atmospheric temperature renders this easy of accomplishment. In the summer, the fulfillment of these conditions involves the use of efficient methods of refrigeration on the part of the dairyman, the transportation company, the milk jobber or handling company, and finally on the part of the consumer to whom the milk is delivered.

It is the preliminary seeding of milk with the noxious germs of filth and their deliberate and luxuriant cultivation by conditions of temperature both of the milk and its surroundings, often simulating that of a laboratory incubator, which is one of the chief causes of the high degree of diarrheal mortality during the summer months of the infants to which it is fed. Instead of the phrase "milk warm from the cow," so common in popular literature, we must substitute the words "milk cold from the cow" and see to it that they become an actuality.

COMPOSITION OF COW'S MILK.

The composition of cow's milk from different breeds varies chiefly in the content of butter fat, the other ingredients being remarkably constant in their proportions. Holstein cattle produce milk with the lowest fat content (3 per cent) and Jersey cattle that with the highest (5 per cent). According to the analyses of Richmond, Fleischmann, United States Experiment Station, Adriance, and others, good herd milk has the following average composition:

Average herd milk of good quality.

	Per cent.
Fat	4.00
Proteids	3.50
Salts	.75
Sugar	4.50
Water	87.25
	100.00

Caloric value per kilogram, 700 calories.

Physical characteristics of cow's milk. Its color is white, varying from a clear to a yellowish white. It is very opaque, the opacity being due to its large content of calcium in combination with its casein. Its specific gravity varies from 1.028 to 1.033, with an average of 1.031. Its taste is pleasant and characteristic. The addition of acetic acid causes a flocculent precipitate, and on the addition of rennet it coagulates into a firm mass.

Reaction. When freshly drawn this is amphoteric or slightly alkaline; on standing it soon becomes acid.

Proteids. The proteids of cow's milk consist mostly of casein in combination with calcium. As in woman's milk, lactalbumen is also present but in small quantity. According to Koenig, casein is present in the proportion of 7 to 1 as compared to lactalbumen.

Fat. The fat in cow's milk is the element that is most subject to variation, as the content of the other food elements is remarkably constant. A milk poor in butter fat contains about 3 per cent, while a rich milk, such as milk from Jersey cows, contains 5 to 5.25 per cent of fat. It is highly important to know the percentage of fat present in milk actually being used for the feeding of infants, for reasons discussed later on.

The sugar of cow's milk is practically identical with that of woman's milk, and is present in the proportion of about 4.50 per cent.

Salts.—Inorganic salts are present in the proportion of 0.75 per cent, of which calcium and phosphoric acid are the most abundant constituents.

Bacteria.—Cow's milk always contains a large number of bacteria, their number increasing with the age of the milk and the conditions under which it is kept.

Cream.—Cream is merely cow's milk rich in fat to excess. It is obtained either by skimming the milk (gravity cream) or is separated from it centrifugally by a machine known as a separator. It differs from milk but slightly in its other solids. Very rich cream (40 per cent) contains relatively less sugar and proteid (sugar 3 per cent, proteid 2.20 per cent). The usual strengths of separated creams marketed contain respectively 8 per cent, 12 per cent, 16 per cent, 20 per cent, and 40 per cent of fat.

PART III.—INFANT FEEDING.

NUTRITIVE REQUIREMENTS OF INFANTS.

It is obvious that any inquiry into the methods of infant feeding demands an intelligent comprehension of their nutritive requirements. Yet this phase of the question has been the object of scientific study only of recent years. Why this has been so, it is difficult to understand, seeing that we have long been possessed of very precise data as to the calorific value of the various articles composing the adult's dietary and the amount of heat units required to maintain their nutrition under various circumstances. And yet, until the investigations of Heubner and his co-workers, our knowledge of the nutritive needs of infants has been mainly empirical and based merely on clinical observations, observations which I may add have been the source of serious error.

The world is indebted to O. Heubner of Berlin, who was the first of a series of investigators, now rapidly increasing, for the discovery of facts which go a long way toward solving the difficult problem of infant feeding. Heubner,[a] appreciating that the principles underlying the feeding of infants, could only be worked out, as in the case of adults, from the logical basis of the number of calories per kilogram of body weight required by them for the purposes of growth and nutrition, undertook a series of exhaustive experiments with this end in view.

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[a] O. Heubner, Die Energiebilanz des Säuglings. Zeitschrift f. diätet. u. physik. Therapie, 1901, Vol. V, p. 13.

Like most fundamental investigations, his line of procedure was quite simple, being merely to determine the daily amounts nursing children took at the breast, each day for successive months, to tabulate the daily and weekly gains observed, and finally to determine by chemical analysis the composition, and from that the calorific energy of the milk that produced it.

His results show that, on the average, nursing infants, in order to thrive, require the following food value, or energy quotient as he calls it, per kilogram of body weight: First week in life, 60 calories[a] per kilogram of body weight; first three months, 100 calories per kilogram of body weight; second three months, 100 to 90 calories per kilogram of body weight; third and fourth three months, 80 calories per kilogram of body weight.

His results were confirmed by Feer, Nordheim, Beutner, Czerny, Keller, and others. He also found that an energy quotient of 70 was the minimum on which a child of less than 1 year of age could maintain its weight. Any diminution of the quotient below this level was followed by a loss. Moreover, the researches of Czerny and Keller[b] have shown that the energy quotient of 100 calories per kilo of body weight marks an upper limit which can only be temporarily surpassed without inducing disastrous nutritive and gastro-intestinal disturbances. These disturbances will be later discussed under the head of "overfeeding."

A necessary part of these researches was the determination of the caloric value of mother's milk. As human milk varies in composition, not only in different individuals but at different stages of its secretion from the breast, only average values could be found. According to the richness of the milk, it varied from 614.2 to 723.9 when lactation was fairly established with an average caloric value of 650 to the kilogram.

The determination of the caloric value of any food substance is very easy once we know its percentage composition as one gram of fat produces 9.3 calories and one gram of proteid, and one gram of carbohydrate have each a caloric value of 4.1.

Thanks to these investigations we are now in a position to determine most exactly, if desired, the amount of food required by each individual child in order to nourish it and give it growth and have, furthermore, data by which we determine whether it is getting too much, in time to avert the disastrous consequences.

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[a] This term, as used in this paper, refers to large calories, or the amount of heat requisite to raise 1 kilogram of water 1° C. in temperature.

[b] Czerny and Keller. Des Kindes Ernährung, Ernährungsstörungen und Ernährungstherapie.

METHODS OF FEEDING INFANTS.

There are three methods by which infants may be fed: (*a*) Maternal nursing, (*b*) mixed feeding, part maternal and part artificial, and (*c*) artificial feeding exclusively.

MATERNAL NURSING.

Importance of maternal nursing.—The importance of maternal nursing can not be overestimated. Were mothers able universally to nurse their children, one-third to one-half of infant deaths would be expunged from our mortality returns.

The number of women capable of nursing their children is probably greater than is supposed. Von Bunge's[a] statistics, gathered from all parts of Europe, tend to show that probably 75 per cent of all women could nurse their children. I have already adverted to the German city of Barmen, where 63 per cent of all infants are fed at the breast. Professor Budin's[b] statistics of the Clinique Tarnier in Paris show that 448 out of 557 women who attended were able to nurse their children. The importance of maternal nursing is well recognized in France, both by the Government and by commercial interests. The effect of this encouragement upon public sentiment has been marked, and in some of the industrial districts of France where formerly the artificial feeding of infants was the rule it has now become the exception.

In most of the factories of that country employing women, notices are conspicuously posted setting forth the advantages of maternal nursing. In many of these establishments rooms are set apart wherein mothers nurse their children, and they can always obtain leave of absence at appropriate intervals for the purpose of suckling their infants.

In Italy a law has been passed compelling each industrial establishment employing 50 or more women to furnish rooms for this purpose.

Causes preventive of maternal nursing.—Three causes are mainly operative in depriving infants of their right to the breast. First, physical inability on the mother's part to nurse her child; second, inability on her part by reason of her engagement in some industrial pursuit; and third, disinclination, chiefly by reason of the trouble maternal nursing involves and the divorce it necessarily entails from social pleasures and pursuits.

Von Bunge has shown that apart from local and systemic disease, alcoholism seems to be the chief cause, in any country as a whole, which renders mothers as a class unable to nurse their children. The daughters of the third generation of alcoholics are usually unable to suckle their young.

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[a] Von Bunge, Die zunehmende Unfähigkeit der Frauen ihrer Kinder zu stillen.

[b] Budin "The Nursling."

The second condition referred to, i. e., the engagement of the mother in some industrial pursuit, depends in a great measure upon the willingness of husbands to accept the earnings of their wives at the expense of their children, or upon their failure to provide for them. This forces the mother to work for her bread while her child is turned over to the tender mercies of some stranger and the milk bottle.

Much can be done by general popular enlightenment to eliminate the third cause, namely, the disinclination of mothers to nurse their children. It is hardly to be supposed that any woman will refuse to nurse her baby from purely selfish considerations, once she is fully informed of the enormous advantages it confers upon her child. It is obviously the duty of the medical profession to further this end by every means at their command.

In view of the foregoing, every mother should nurse her child unless there are cogent reasons to the contrary. The following causes may be mentioned as contraindicating maternal nursing:

(1) Tuberculosis, latent or active, affecting the mother. By nursing the child she can but accelerate the progress of the disease, besides exposing the child to the danger of contracting it.

(2) When the mother is affected by grave, chronic, or systemic disease.

(3) When the mother is choreic or epileptic.

(4) If she has suffered from any severe complication of the parturient state, such as hemorrhage, eclampsia, nephritis, puerperal septicæmia, and the like.

(5) Local disease of the mammary gland.

(6) When as the result of two previous experiences under favorable conditions she has shown her inability to nurse her child (Holt).

(7) When no milk is secreted.

Care of the breasts during lactation.—In order to prevent local affections, both of the mammary gland and the infant's mouth, it is highly important that particular attention should be paid to cleanliness. The nipples and breasts should, therefore, be carefully washed prior and subsequent to nursing, either with plain water or boric-acid solution.

Nursing during childbed. A newborn child should be nursed once in six hours the day following delivery and once in four hours the succeeding day. This is necessary (1) to accustom the child to take and the mother to give the breast, (2) to empty the breasts of colostrum, (3) to promote the involution of the uterus.

The colostrum furnishes the child with all the calories necessary to its needs until the lacteal flow is established, nor does it need any other food. The usual practice of feeding a newborn child with decoctions of various sorts with the object of alleviating the colic supposedly indicated by its cries is absolutely to be prohibited. The crying promotes the full expansion of the lungs and the establishment of normal circulation. Plain boiled water, however, may be freely given, as the body fluids of the newborn are in a concentrated state.

Regular habits of nursing. Much more is dependent upon the early establishment of regular nursing habits than is supposed. They are as easy to initiate as the irregular, and by so doing much of the strain of lactation upon the mother can be eliminated. This is highly desirable in view of the beneficial effect a calm and equable state of mind and adequate time for rest and sleep have upon the lacteal secretion. Moreover, the milk is more likely to be of a uniform character throughout lactation when the breasts are drawn upon at intervals definitely spaced. A young infant can usually be expected to take a long nap of some four or five hours during some period of the twenty four, and it is just as easy to have this secured at night. In order to promote the uniformity of lacteal secretion necessary to regular growth, the intervals between nursing should not be too short. Nor, on the other hand, in the earlier months at least, should they be too long. In the first instance, the milk is apt to be too rich and concentrated (Rotch), thus causing overfeeding, and in the second, it is apt to be deficient in nutritive elements. Huebner is of the opinion that the number of nursings in the twenty four hours usually advocated is too great, and a less number is productive of better results. In this view he is confirmed by Czerny, Keller, and others.

The following schedule seems to fulfill the best practice in this direction:

Age	Number of nursings in 24 hours	Interval during day	Night
First day	4	6	1
Second day	6	4	1
Third to twenty eighth day ..	8	2½	1
Fourth to thirteenth week .	7	3	1
Third to fifth month	6	3	0
Fifth to twelfth month	5	4	0

It may not always be possible to carry this schedule into effect, but every effort should be made to do so. In the majority of cases, however, no difficulty will be experienced, provided the child be always awakened, if necessary, when the time comes to put it to the breast. Regularity in nursing intervals is of great assistance to the mother in providing for her adequate recuperation, and the unbroken sleep at night permits her to continue lactation long after the time she would otherwise have to abandon it.

Mode of giving the child the breast.—It is surprising to note the ignorance of some mothers even in such essential details as this. It is, therefore, necessary to see that the breast is properly presented to the child. The child should be held in such a position that it can seize squarely upon the nipple, which should not be presented obliquely to it. It is important to avoid pressing the child's nose into the breast, in order to allow it free respiration.

Signs of successful breast feeding.—The child who is receiving adequate nourishment from the breast performs all its functions with the optimum of regularity. Its sleep is peaceful, its appetite is keen, and it presents a general appearance of contentment and bien être. The bowel movements are free, and consist of two or three golden, smooth, salve-like discharges a day. The urine is odorless, limpid and adequate in amount. The body tissues are firm and elastic, and their outlines plump and rounded. Most important of all, the child gains steadily and constantly in weight. If an infant cease to gain in weight something is always wrong. On the other hand, sudden and abnormal gains in weight point to excessive overfeeding, and require a reduction of the nourishment in amount.

After the initial loss following birth has been regained, the average healthy child increases in weight during the first three months from 120 to 150 grams (4 to 5 ounces) every week and from the third to the sixth month from 100 to 120 grams (3½ to 4 ounces). The effect of these increases is to double the initial weight at six months, and to treble it at the close of the first year. Large children gain absolutely but not relatively greater weekly amounts than the small. This rate of gain may be considerably increased within physiological limits, the main indication of abnormality being a wide departure from a rate of gain previously observed. To this end infants should be regularly weighed on sets of scales sensitive to 15 grams (½ ounce). A careful record of the weight should be kept.

Signs of inadequate nursing.—The early detection and diagnosis of abnormalities in the quality and quantity of the milk furnished by the mother is imperative; for, unless corrected, they are fraught with danger to the child. While nothing can take the place of good natural nursing, and while poor artificial feeding is the very worst method of infant nutrition we have, poor maternal nursing is an inferior alternative to good artificial feeding. We should, therefore, endeavor to discover the difficulty as soon as possible in order that hasty weaning of the child should not take place when the symptoms are unconnected with the food, or the indigestion from which it is suffering is due to causes temporary or remediable. On the other hand it is obvious that, if from every symptom maternal nursing is going to fail, it should not be allowed to continue because the mother desires it from mistaken notions of her duty to the child.

Inadequate nursing, insufficiency.—During the first few days of life the temperature of the child furnishes a very important indication, not so much of the nourishment as of the amount of fluid it is receiving. Very few children who are receiving a sufficient quantity of fluid from the breast during the first few days of life present abnormalities in the temperature.

Fever of inanition.—Many of those who get little or nothing during this time have an elevation of temperature of 38.4 to 39.1 C. (101 to 102 F.), while in exceptional cases the temperature rises to 40 or even 41 C. If no other obvious symptoms of disease are present, such a temperature observed on the second to the fourth day may be considered evidence of insufficient ingestion of fluid or even of starvation. Supplying the needs of the infant in this respect rapidly causes a disappearance of the fever.

If the milk of the mother's breast be insufficient to supply a greater energy quotient than 70, the child ceases to gain in weight; if below this, a loss ensues. The child is fretful and seems always hungry, as indicated by continuous sucking of the fingers and remaining a long time (forty or fifty minutes) at the breast. If it stops then, it is rather from exhaustion than because it is satisfied. If the insufficiency of food has been very great from the outset, it may lie in a remarkable condition of apathy (Budin), sleeping most of the time. Weighing the child before and after nursing shows that it gets very little. The mother's breasts are not full and tense at nursing time, as they should be, and during the intervals of feeding but little milk is present in them. The child's discharges, both fecal and urinary, are very small, and, most important, there is a steady loss in weight.

Inadequacy of maternal nursing (overfeeding).—This is not so frequently seen in children at the breast as in those artificially fed. It is mainly observed in the case of strong, full-blooded mothers or wet nurses, whose milk from rich food and insufficient exercise is highly charged with fat. Overfeeding is briefly characterized by the following symptoms, the cause of which will be discussed in another connection: They are irritability, restlessness, and broken sleep, followed by constipation with gray, dry stools. The urine is odorous and stains the diaper. A continuance of the cause finally induces severe gastro intestinal symptoms: vomiting and diarrhea are present, with discharges containing curdy masses of fatty soaps. The gain in weight diminishes, ceases, or a loss is manifest.

Value of the examination of the breast milk when the infant is not "doing well."—In many cases when the child is not thriving an examination of the breast milk may give valuable information. I have given elsewhere clinical methods by which an approximate idea of its qualities may be obtained. The result of the milk examination usually discloses (1) that it is too rich in quality and unusually abundant in quantity; (2) that it is scanty and poor in quality; (3) that it is abundant, but poor in quality.

Over-rich milk.—I have already adverted to this condition and the gastro-intestinal disturbances it may induce. Clinically, over-rich milk is indicated by a high specific gravity (1.032 to 1.036), combined with a high percentage of fat. The child should not be weaned, but the quality of the milk regulated by less frequent nursings, a simple diet, and exercise in the open air to the point of moderate fatigue. As the symptoms in the child are caused by the continuous ingestion of a food far superior in caloric value to his nutritive needs, the diminished frequency of the nursings not only enables it successfully to rid itself of the excess of nutrient elements ingested, but also tends to diminish the richness of milk secreted.

Scanty milk of a poor quality. -This condition is most frequently manifest in delicate and anæmic mothers. The amount present in the breast may be so small that the small quantity of milk necessary to make the examination is secured with difficulty. The clinical characteristics of this milk are low specific gravity (1.024 to 1.027) and cream only 2 to 4 per cent. In such cases the quality of the milk is so poor and the quantity so small that it is manifestly impossible to nourish the child by it.

In other instances the variation from the normal is not so great; i. e., specific gravity 1.028 to 1.030, cream 4 per cent, with fairly abundant quantity. In such cases we may hope to improve the quality of the milk by appropriate measures. These are adequate rest for the mother at night, fresh air, nourishing food, and gentle exercise. The anaemia usually present may be improved by malt extracts and preparations of iron. If this condition is dependent upon conditions incidental to the lying-in period, the outlook is usually good. If, on the other hand, it is the result of constitutional debility or neurotic diathesis, improvement may only be temporary, necessitating the weaning of the child, not only in its own interests, but those of the mother as well.

Abundant milk of poor quality. -This condition is sometimes seen in anaemic subjects who have been taking large quantities of malt or alcoholic beverages in the hope of improving their milk supply. In such cases the quantity may be so abundant that the breasts are painfully full at nursing times and between them may flow away spontaneously. Clinical examination shows low specific gravity and very low fat.

Owing to the grade of hydremia accompanying these conditions, very little improvement can be expected to ensue from treatment, and nursing should be discontinued.

Wet nursing. The milk of another healthy woman is the best substitute for maternal nursing when the mother's supply has failed. Expense and the difficulty of obtaining a good wet nurse in this country are countervailing circumstances. The wet nurse should be a perfectly healthy woman free from syphilis or tuberculosis. A careful physical examination should be made to ascertain these points. Her breasts should be well developed and at nursing time be tense and full. A good amount of glandular tissue should be present in the breast and pains taken to be assured of that point. The nipple should be of normal development and free from cracks or fissures. Her blood should be rich, as it is impossible for an anæmic woman to give good milk.

Contrary to what is usually supposed, it is by no means necessary that her milk should correspond in age to that of the child. She should, however, have been a nursing mother long enough to be able to demonstrate by the condition of her child that she has plenty of good, nutritious milk. To this end her child should always be inspected before her acceptance. When accepted she should be given a fair chance to adjust herself to her novel surroundings before deciding her incompetent as a nurse, as these conditions may, at first, notably affect her supply of milk.

Weaning. The time at which weaning should take place is subject to considerable variations. With an abundant supply, nursing may usually be continued to advantage during nine or ten months of the first year. Some mothers are able to extend this period through the twelfth month. After that time breast feeding is seldom advisable. As a usual thing, the ninth month marks the time when the breast must be supplemented by other food.

Method of weaning. Weaning should be gradually done, both in the interests of the mother and the child. Much of the difficulty attending the gradual weaning of infants may be eliminated by accustoming the child to take a bottle from the outset. This may be accomplished by feeding it boiled water from a bottle from the beginning. In the absence of such measures the experience is too frequent that the child refuses all other food as long as it has access to the breast, and nothing short of actual starvation will induce it to accept either the bottle or the spoon.

Weaning during the hot months should be postponed if possible; but the harm thus done is not usually so great as the continuance of the child on an inadequate breast milk.

Indications for sudden weaning. Sudden weaning may be necessary at any time on account of the development in the mother of severe acute disease, such as typhoid fever or pneumonia; of grave systemic disorders, as tuberculosis or nephritis; from the intercurrence of pregnancy or of disease of the mammary gland. Minor illnesses or acute sickness of short duration are not indications for weaning, though if the attack be severe the infant may be placed temporarily on the bottle, and the flow of milk maintained by the breast pump. Upon the establishment of convalescence, the infant is returned to the breast.

Mixed feeding.—This is a combination of breast and artificial feeding. It is useful when the mother's milk is good but somewhat deficient in quantity. It may also be adopted when it is evident that the strain of maternal nursing is making inroads on the mother's reserve of health. She may then be relieved to the extent of one or two nursings a day, and thus considerably prolong the period of her lactation. Mixed feeding is also useful as an expedient to bridge over temporary insufficiency in the mother's supply of milk. It is not expedient, however, to reduce the maternal nursings by three or more a day, lest the mother suffer a serious deterioration in the quality of her milk.

ARTIFICIAL FEEDING.

In considering the artificial feeding of infants, there are several general principles which have received universal acceptance:

First. That as mother's milk is an ideal food, supplying the infant with proteid, fat, and carbohydrate in proportions adapted to its needs, the only logical substitute is a food that will do the same.

Second. The substitute should approximate mother's milk (*a*) in the energy quotient that it furnishes, (*b*) in proximate principles, and (*c*) in the amounts necessary to maintain nutrition.

Third. These conditions are secured only by some animal milk.

While infants have been sucessfully fed on the milk of other domestic animals, such as the goat, the horse, and the ass, cow's milk is the only substitute commercially and practically available in this country as a food for the artificial feeding of infants less than 1 year of age.

Comparison of cow's milk and woman's milk. In order to proceed with intelligence it is necessary to compare the average composition of woman's milk with cow's milk. The following table shows the difference between their average composition:

	Woman's milk.	Cow's milk.
	Per cent.	*Per cent.*
Fat	4.00	3.00
Proteid	1.50	[illegible]
Sugar	7.00	[illegible]
Salts	.20	[illegible]
Water	[illegible]	87.25
	100.00	100.00
Calories per kilo	710.50	[illegible]

Average.

Cow's milk is more opaque than woman's milk, although woman's milk may contain a greater percentage of fat. This is due to the greater content of calcium salts in cow's milk by reason of its greater proportion of casein.

In reaction cow's milk, though slightly alkaline or amphoteric when freshly drawn, soon becomes somewhat acid, while woman's milk is amphoteric or alkaline.

As there is very little difference in total solids between the two, their specific gravity is about the same.

The sugar of cow's milk and woman's milk is lactose in complete solution. They differ, however, in quantity, as woman's milk contains 6 to 7 per cent, while cow's milk has usually 4.5 per cent. The greater part of the fat in cow's milk is neutral fat, as in woman's milk; but cow's milk contains far greater quantities of the volatile fatty acids, of which there are but traces in woman's milk. It is in the proteids that the chief difference between cow's milk and woman's milk is manifest. Cow's milk has on the average 3.50 per cent of proteid to 1.50 per cent in woman's milk. The reason for this difference is obvious. As all growth is dependent upon proteid material, and animals are unable to build up proteids within themselves from the nonnitrogenous portions of their diet, they are dependent for their supply upon the ingestion of proteid in their food. As the ratio of growth of the calf compared to the infant is about as 2 is to 1, it follows that the calf requires relatively twice as much proteid as does the infant. Moreover, owing to differences in their respective digestive tracts, the proteid in cow's milk is of a different composition from that of woman's milk. In the human being the stomach forms 20 per cent of the intestinal tube and digestion is chiefly intestinal. In the cow the stomach forms 70 per cent of the digestive tract and digestion is chiefly gastric. Under the action of the active rennet ferment present in the stomach of the calf, cow's milk forms a large curd which remains in the calf's stomach until digestion is complete. In the infant the soft, flocculent curd of mother's milk is adapted to easy transit from the stomach to the duodenum, and it is altogether likely that a portion of the milk ingested does so pass out before the nursing is finished.

In view of the foregoing, as casein is the curding proteid in milks, we should expect to find, as is actually the case, that the proteid of cow's milk is richer in casein than the proteid of human milk. Koenig gives the following composition of the proteids in cow's milk and human milk:

	Lactalbumen	Casein.
	Per cent.	*Per cent.*
Human milk	1.26	1.03
Cow's milk	.53	3.02

The proteid of cow's milk when coagulated by rennet in the test tube gives a firm, tough, contractile curd. Heubner, however, has shown that we can by no means infer that this is the action in the human stomach; for, if rennet is added to cow's milk in a test tube, the tube subsequently corked, and then slowly turned over end for end to imitate the movements of peristalsis, the resulting curd is every bit as fine as the curd of human milk. This statement Heubner confirmed by withdrawing cow's milk shortly after ingestion by infants with a stomach tube.

This brings us to the consideration of a fact due to the researches of Heubner, Keller, and Czerny, which tends to revolutionize all our preconceived notions on this side of the Atlantic at least as to the digestibility of cow's milk proteid. For many years it has been held that the proteids of cow's milk are very difficult of digestion. To overcome this supposed difficulty very many devices have been advocated. Thus various alkalies and diluents have been applied, the percentage of proteids has by modification been attenuated almost to the vanishing point, split proteids have been advocated, a portion of the casein being replaced by whey proteid—in short, almost every conceivable device that ingenuity could suggest.

This would be highly commendable were cow's milk proteid really so difficult of digestion by the human infant. The researches of the investigators just adverted to have shown this important fact, viz. that cow's milk proteid is almost as easily digestible per se by infants as are the proteids of woman's milk. In this country Brennemann and Walls[a] have confirmed this view. On the other hand, Czerny and Keller have shown that the element in cow's milk which causes digestive disturbance is the fat and not the proteid. They have given us a very precise and definite clinical picture of these disturbances, a picture perfectly familiar to all who have dealt much with the artificial feeding of infants, but which has been ascribed heretofore in this country to difficult proteid digestion.

Let us then examine the basis for this belief which has hitherto been regarded as a fundamental fact ever to be considered in the percentage system of the modification of cow's milk. It has been based for the most part on these facts: First, that in the stools following gastro-intestinal disturbances in infants a large number of apparent curds are mingled with the fecal discharge. They look like curds and have been taken for curds without further investigation. Czerny has shown that they consist for the most part of saponified fat, neutral fat, and fatty acids, interspersed in severe cases with clumps of bacteria.

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[a] Am. Journal Med. Ass., 1907, Vol. XLVIII, 1338 1344; Ibid., F. X. Walls, pp. 1389 to 1392.

If infants are fed on fat-free cow's milk, although the milk be undiluted and containing 3.50 per cent of bovine proteid, no trace of casein appears microscopically in the stools, yet Chapin tells us it is rare to find an infant 10 months of age who will digest more than 1.50 to 2 per cent of cow's milk proteid.

In this country, the digestibility of cow's milk proteid has been confirmed by Walls[a] after a series of hundreds of observations. Now Czerny has shown that the persistent ingestion in a child of a diet with an energy quotient surpassing 100 calories per kilogram of body weight is invariably followed sooner or later by nutritional disturbances. Owing to the fact that infants receive for the most part nothing but milk, and that the percentage of sugar and proteid in milk is pretty nearly constant, it follows by reason of the high caloric value of the fat (9.3 calories) that a high energy, quotient implies an excess of fat. It is impossible, by mere inadvertence, with carbohydrate or proteid alone to get an energy quotient of a dangerous height without producing a food which either from its state of concentration or by reason of its bulk would be obviously unfit to feed to any infant. On the other hand, slight increases of 1 or 2 per cent in the fat content of a food may have the effect of raising its caloric value to a dangerous extent.

The action of an excess of fat in causing nutritional disturbances in infants operates in two ways; first, by reason of its action on the alkaline bases of the body, and second, by its influence on gastric digestion.

Action of excess of fat on alkaline bases of body.—According to Czerny and Keller, the deleterious influence of an excess of fat in the diet is usually operative by reason of the abstraction such excess entails on the alkaline bases of the organism for the purposes of its saponification in the intestinal tract. It is evident that, owing to its high caloric efficiency and the greater difficulty of its oxidation as compared both with proteid and carbohydrate, the general capacity of the organism for the absorption of fat is strictly confined within narrow limits. Whereas an excess of proteids and carbohydrates is disposed of rather easily by the process of metabolism, with fats such is not the case. An excess of fatty food is not absorbed, but remains in the intestine and is there saponified. This is not due so much to efforts of the organism to get rid of the fat in this way as to the natural tendency of fat to undergo this action when exposed to the action of alkaline fluids such as the intestinal secretions. In this way an excessive abstraction of the alkaline bases of the body takes place, which are in turn supplied to the body, in milk at least, in quantities merely sufficient for a normal diet. The income, then, of these bases becomes less than the outgo, and a pathological condition due to this diminution is thereby induced. Owing to the greater energy required in its digestion, the tendency of cow's milk is to remain longer in the intestinal tract. This gives greater opportunity for any excess of fat present to rob the body of alkaline bases by virtue of its saponification. As a consequence of the general richness of cow's milk in this country, such danger of excess of fat must always be present when the milk used as an article of infant food is not controlled in this respect. It would seem that a milk which contains 4 per cent of butter fat were somewhat too rich and that a fat content of 3 to 3.5 per cent would be nearer the mark to insure success in infant feeding. The experience of dairymen tells us that calves do best on this; moreover, the production of rich milk in cows is not attended by a corresponding increase in the salts present, as rich milk is a result of careful breeding for that purpose by man, and is not a condition original to the milk of the cow.

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[a] F. X. Walls, Am. Jour. Med. Sci., 1906, II.

The second effect excessive fat has in promoting gastro-intestinal disturbances lies in its behavior when casein is curded by rennet in the stomach. It is well known, among cheese makers at least, that casein, when coagulated by rennet, carries down with it a very large per cent of the butter fat present. This is well shown in the case of the cream cheeses, some of which contain nearly six times as much fat in their composition as casein. As the whey from whole milk contains no casein and less than 1 per cent of fat, it is obvious that the casein when curded brought down with it some 75 per cent or more, according to the richness of the milk, of all the butter fat originally present in the milk. The practical bearing this has upon the digestibility of cow's milk proteid is as follows: The richer the milk in fat, the greater percentage of fat will then be in the curd formed by the gastric digestion.

A very simple experiment will show in what way the digestibility of a rich whole milk would thereby be affected. Place a small portion of skimmed milk in a small flask with a narrow neck. In another flask place a similar portion of a rich milk containing say 5 or 5.25 per cent of fat. Add a small portion of liquid rennet to each, cork, and turn them slowly over and over in order to imitate peristaltic action. In the case of the skimmed milk, as curdling takes place, we see the formation of a flocculent curd, each particle of which remains separate. In the case of the milk rich in fat, the curds are dense and heavy, and show a remarkable tendency to coalesce and stick together. This is well brought out by attempting to pour out the curded milk from each bottle. Now, the curded skimmed milk pours out very easily, and, on examination, the curds are distributed throughout the whey in light flocculent masses, while in the case of the milk rich in fat, pouring out will frequently be interrupted by large lumpy curds sticking in the narrow neck. Agitation of the bottle to break them up merely seems to increase their adhesive powers, each lump receiving further accretions from the particles floating in the neighborhood.

We can easily, then, predict the events that take place when a milk rich in fat is acted upon by the gastric juices in the infant's stomach. In fact, this action of fat in making curds, large and indigestible by reason of their excessive fat content, has long been known to dairymen, as they are well aware of the fact that Jersey cows often can not nurse their calves by reason of the excessive richness of their milk. And yet one would never for one moment suppose, under normal circumstances, that the casein of cow's milk is ever indigestible *per se* to the calves for which it is intended.

In the case of the human infant, if the milk be too rich it is vomited. If it is just rich enough to produce a curd with a fat content greater than it should have, peristalsis is checked and the stomach discharges its contents slowly. This retarded action in the discharging action of the stomach is to permit its gastric juices to act upon the casein in the fatty curds present. As a result, the next meal finds the stomach with a residue from the one previous, to which is added the increment just received. In this manner the gastric contents become progressively richer both in fat and proteids. As this highly seasoned mass is poured out little by little into the duodenum and small intestine, it, in its turn, becomes encumbered with a food the problem of whose digestibility is beyond its resources. Gastric as well as intestinal digestion is well-nigh at a standstill; fermentative changes take place, and then, in the language of the German investigators, the so-called "'catastrophe' ensues."

This, of course, is an extreme case. Czerny has graphically delineated the symptom complex observed when the feeding of foods is persisted in whose energy quotients surpass the normal limit of the organism, especially when such excess consists of fat. An infant that has been thriving receives some new addition to its food. This may either be an increase in the quantity or an addition to the richness of its ingredients. For a while a remarkable gain in weight is observed. The infant, however, soon becomes restless, its sleep is light and broken. It seems somewhat nervous and becomes less active and playful. At the same time its keen appetite diminishes and it has to be coaxed to drink its food. Often 2 or 3 ounces will be left in the bottle or glass. The stools, hitherto of normal odor, color, and consistency, become pale-gray, hard, and dry. In fact, they are of the color and consistency of putty, and may be rolled off the diaper without even soiling it. Their odor is strong and suggests decomposition. The urine becomes charged with ammonia salts and stains the diapers. Systemic effects are shown by the pallor of the child; the tissues lose their firmness and solidity, becoming flabby and relaxed; the child also fails to gain in weight on the same food or a greater quantity of the same food which has hitherto produced a gain in weight. A persistence in this course of feeding not only does not increase the weight, but occasions a loss. Two results follow the continued exhibition of such a diet. The body tissues waste, the belly distends with gas, and we have the atrophic or marantic child, or a gastro-intestinal catastrophe results with vomiting, diarrhea, fever, and prostration, accompanied by an inability to take cow's milk for a period often extending into weeks. Such, then, are the symptoms in the more aggravated cases. In those more chronic the anorexia and constipation go hand in hand, the former and the latter being almost constant. Outbreaks of eczema are common, from small roughened patches on the cheeks to eruptions invading large areas. As the child grows older the symptoms of rachitis are observed.

The firm, pale-gray stools so characteristic of this condition are composed largely of fatty soaps (Seifenstuhlen). Czerny and Keller ascribe the pathology of this condition to the action of fat in extracting the alkaline bases already alluded to, as shown by the increased elimination of ammonia in the urine. They regard this elimination of ammonia salts due to the fact that the alkaline bases are so largely drawn upon from the body to saponify the excessive amount of fat accumulated above the needs of the organism in the intestine, that in order to satisfy the normal acid products of metabolism the ammonia salts are drawn upon.

Percentage system of artificial feeding.—In this manner we see divergent points in this view of the whole question of infant feeding as compared to that in vogue on this side of the Atlantic. The percentage system of modification of milk rests upon the following premises, two of which are certainly faulty:

First. That a substitute for mother's milk must resemble it in the relation and chemical composition of its ingredients;

Second. That this condition is fulfilled only by some milk;

Third. That cow's milk is the only practical substitute;

Fourth. That the chief difficulty in the use of cow's milk is the indigestibility of its proteid by infants; and, consequently,

Fifth. The composition of cow's milk must be so altered by appropriate manipulation as to provide those relative proportions of proteid, fat, and milk-sugar which experience teaches us is suitable for the different ages of the child.

We will now proceed to discuss wherein these principles are erroneous. The error seems to be embodied in principle 4 and principle 5.

The fourth principle, i. e., that the casein of cow's milk is indigestible, has been shown to rest upon a misconception. It only helps in a mechanical manner to produce indigestion when combined with large quantities of fat, or is altered in its digestive quality by fermentative changes in the milk at the time of consumption.

It is the taking of the assumption of the indigestibility of the casein of cow's milk for granted that has caused the rather numerous instances of overfeeding in the case of American children. In fact, under our present system overfeeding is apt to be the rule rather than the exception. By reason of the belief that the proteid of cow's milk is the essential indigestible portion of its ingredients, it has been reduced, in the milk modifications prescribed by many of our physicians, almost to the point of extinction in certain individual cases.

Realizing, however, that food offered to a child must contain certain elements of energy, the place of the proteid has been supplied by the use of fat, which, until in recent years, has been regarded digestible by many of our pediatrists and to act in conjunction with the carbo-hydrates as a proteid saver, besides promoting free action of the bowels.

And yet what better proof that fat is the cause of constipation could be adduced than by the following circumstance which Walls[a] has observed in so many instances, viz, that whenever the symptoms of fat intoxication are manifest, as evinced by the characteristic stools, a diet of fat-free whole milk works like a specific in initiating normal intestinal action.

The fifth principle, going hand in hand with the fourth, is likewise erroneous in basing the composition of the milk modifications offered on the age alone of the child who receives them in so far as the weight of the child and the amount of the heat units necessary to nourish it and give it growth are not taken into proper consideration thereby. Moreover, only lately has the principle been recognized that food elements, if furnished even in trifling excess, instead of inuring to the benefit of the infant, have a way of reacting to his detriment. The following table, taken from a well-known text-book on pediatrics, can be used to further illustrate these points:

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[a] Loc. cit.

Approximate schedule for milk modifications for various ages.

Age	Fat	Sugar.	Proteid.	Quantity at one feeding.		Number of feedings in 24 hours.
				Ounces.	Grams.	
Premature infants....	1.00	4.00	0.25	$\frac{1}{4}$ – $\frac{3}{4}$	7–22	12–18
First to fourth day....	1.00	5.00	.30	1 – 1½	30– 45	6–10
Fifth to seventh day.	1.50	5.00	.50	1 – 2	30– 60	10
Second week....	2.00	6.00	.60	2 – 2½	60– 75	10
Third week....	2.50	6.00	.80	2 – 3½	60–110	10
Fourth to eighth week....	3.00	6.00	1.00	2½ – 4	75–125	9
Third month....	3.00	6.00	1.25	3 – 5	90–155	8
Fourth month....	3.50	7.00	1.50	3½ – 5½	110–170	7
Fifth month....	3.50	7.00	1.75	4 – 6	125–185	7
Sixth to tenth month....	4.00	7.00	2.00	5 – 8	155–250	6
Eleventh month....	4.00	5.00	3.00	7 – 9	220–280	5
Twelfth month....	4.00	5.00	3.00	7 – 9	220–280	5
Thirteenth month....	4.00	4.00	3.50	7 –10	220–310	5

If, for example, we take from this table the formula advised for the second week—i. e., fat 2.00, sugar 6.00, proteids 0.60 per cent—we find such a ration furnishes a caloric value of 0.457 calories per gram. The total amount administered is 600 to 750 cubic centimeters a day, giving a caloric value of 274.2 to 342.75 calories per diem. Assuming that a healthy average child weighs 3,500 grams at the beginning of the second week, the energy quotient required is 100 calories per kilogram of body weight. It will then require food supplying 350 calories a day properly to nourish it.

We see, however, that not only the lowest but the highest ration furnished by this table is less than its actual requirements. The ration is also very poor in proteid in contradistinction to mother's milk, which is normally higher in proteids during the first weeks than at other periods of lactation. Of course, the theory for this modification is that higher proteid would upset the digestion. This objection can be no longer regarded as valid. This is not, however, a very serious disadvantage of this table. Under-feeding never caused serious gastro-enteritis, and as a consequence babies fed on this plan usually pass without digestive disturbance through the initial formulæ, though they gain weight but slowly from them.

It is when we turn to the percentages advised for older infants that the possibilities of danger are apparent. Taking the daily ration advocated for the infant of 6 months, we find that the percentages of this ration are as follows: Fat 4.00, sugar 7.00, proteid 2.00 per cent. This gives a caloric value of 741 calories per kilogram, or 0.741 calories per gram. The daily quantity recommended varies from 930 to 1,500 grams, giving a total daily caloric value of 689.1 to 1,011.5 calories. Supposing a 6-months-old infant to have attained the weight of

7,300 grams (16 pounds), and requiring an energy quotient of 90, we see that the energy quotient furnished by the above diet varies from 92, or near the safety limit, to the dangerous figure of 134.8.

Now, as long as everything is going well, and we have no scientific guide on which to base our calculations, the tendency is to give an artificially fed infant as much food as it will absorb within the indicated limits of the schedule.

As the higher limit of 1,500 grams a day was being approached, a gratifying and remarkable increase in the gain in weight would be observed. This, as Czerny tells us, is a significant sign that the income and the outgo are not correctly balanced and calls for immediate reduction in the energy quotient of the food.

That this position is sound a little reflection will make clear, as it is evident that growth, being the result of anabolic effort on the part of certain organs, must necessarily have a certain maximum rate of increment beyond which we pass into the pathologic. In our hypothetical instance, satisfaction at the child's remarkable and unexpected gain in weight would further the inclination to continue with greater quantities of the food that has agreed so well with it. In this manner the physiological needs of the child having been long surpassed, the symptoms of over-feeding as previously described become evident, either the gastro-intestinal catastrophe or the anorexia, constipation, and continual recession in weight.

In the case of the first outcome, withholding of the milk and appropriate treatment is instituted, with the result, however, that much valuable ground is lost, and we have a child who perhaps for weeks can not take any form of cow's milk at all.

In the second, the results may vary. The digestive disturbances are, as a usual thing, erroneously attributed to the cow's milk proteids. The constipation may be regarded as the result of a lack of fat, which may be added to a diet already overcharged with this, and a gastro-intestinal crisis definitely precipitated.

We must, therefore, regard the percentage system of feeding as based upon conclusions that are incomplete and the result of clinical observation alone, a form of observation that is prone to lead to inferences incorrectly grounded in fact. Without deprecating the skill and care used in working out these formulæ there is occasion for regret that until very lately, in the belief of the complete scientific accuracy of our methods, we have failed to undertake any observations for ourselves as to the nutritive requirements of infants, and the actual digestive absorption that takes place in respect to the various constituents of human and other milks in the infantile digestive tube. We see by the foregoing that the whole question of the artificial feeding of infants may be reduced from a condition of extreme complexity, based upon the incomplete data of clinical observations, to a relatively simple and scientific basis. The principles, briefly stated, consist in feeding to every child who must be artificially fed a food which, based on the age and weight of the child, will furnish it with the energy quotient it requires and no more, and to continue the food at that composition and quantity until a diminution in the weekly gain of weight, unaccompanied by symptoms referable to the gastro-intestinal tract, informs us that an increase in the diet is necessary.

As we need no longer be deterred by our fears of the indigestibility of cow's milk proteid, in the absence of excessive quantities of fat, or changes due to fermentation, undiluted cow's milk can be given from the beginning of the first month on, provided its content of fat does not exceed 2 to 3 per cent, nor the daily quantity greater than 150 cubic centimeters per kilogram of body weight.

To settle any doubt as to the digestibility of whole cow's milk in the stomach of infants, we have but to turn to the observations of Budin, Oppenheimer, Variot, Comby, Lazard, Drapier, Ruflie, Bonifas, Gillet, and others, men of large experience, who have fed infants from the earliest days in life on whole cow's milk in proportion to their needs without observing anything but the happiest results.[a]

In feeding cow's milk undiluted to infants in this country our practice in this direction must be controlled by the following circumstance: Abroad, owing to different methods in feeding, and different grades of cattle, milk containing over 3.75 per cent of butter fat is rarely found, and the average is probably not over 3 to 3.50 per cent in the majority of cases. In our country it is a milk poor in butter fat indeed which does not average 4 per cent, while selected milk supplied to many of our institutions and hospitals and our "certified" grades of milk are often nearer 4.50 or 4.75 per cent. Thus Chapin found that the average content of fat of the milk used in the Babies' Ward of the Postgraduate Hospital in New York was 4.40 per cent. The use of milk of this grade of richness is likely to be followed by digestive disturbances and symptoms of overfeeding when fed whole to infants, both by reason of excessive caloric value and of the formation of large fat-containing curds.

While agreeing with the experience in France of the digestibility of undiluted cow's milk as an infant food, I am not prepared to advocate its use in this country unless the fat content is known to be no higher than 3.00 per cent. This condition can, however, be secured either by using milk from Holstein cattle, which is normally no richer than this, or by removing appropriate amounts of the "top milk" from bottled milk after the cream has risen and then thoroughly mixing the remainder.

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[a] Budin: The Nursling. Comby: Medicine Moderne, Mar. 14, 1894. Lazard: Journal de Cliniques et de Therapeutiques Infantiles, 1,895,886. Darpier: Rapport sur le fonctionnence de la creche. Ruflie: La Gouttette ou la diarrhee verte des nourissons et son traitement par le lait sterilisé. Bonifas: Le Progrès Medicale. Gillet: Formulaire d'Hygiene Infantile Individuelle.

GENERAL DIRECTIONS FOR THE ARTIFICIAL FEEDING OF INFANTS.

For children one month old or over.—First, weigh the child. Allow a daily quantity of cow's milk of one-seventh body weight for infants up to 3 months of age, one-eighth the body weight from 3 months to 6 months, and after that from one-ninth to one-tenth.

Quality of milk to be used.—Use nothing but clean, fresh, bottled milk, "certified" if possible. If this can not be had, use bottled milk from a high-grade dairy, making sure that the fat content does not exceed 3.00 per cent. If it is greater than this it must be reduced to this figure by dipping the cream out of the top of the bottle in the amounts given in the following table and then mixing thoroughly the remainder.

NOTE. A cow's milk of this percentage of fat –3.50 –has a heat value of 653.5 calories per kilogram, or about the average caloric value of woman's milk.

Table showing quantities of top milk that must be removed from top of quart bottles of milk in order to reduce the percentage of fat to 3.00 per cent.

Original percentage of fat in the milk.	Quantity of cream to be removed from top of quart after cream has risen to reduce fat to 3.00 per cent.
4.00	50 cubic centimeters—1⅔ ounces
4.50	75 cubic centimeters—2½ ounces
5.00	100 cubic centimeters—3⅓ ounces

Mix the milk thoroughly by pouring into another vessel and measure out the amount of the daily supply requisite as indicated by the age and weight of the child; e. g.: Weight of child 1 month old, 4 kilograms (9 lbs.); $\frac{1}{7}$ of body weight = 570 grams (19 ounces); daily quantity of milk 570 grams (19 ounces). Divide the quantity of milk so obtained in nursing bottles each containing equal amounts according to the daily number of feedings advocated in another part of this paper (see breast feeding). Sterilize it by standing the bottles, each corked with absorbent cotton, in boiling water up to their necks and boil for a period of three-fourths to one hour. Cool and preserve the bottles on ice until required. Before feeding heat the milk to blood temperature by standing the bottle in hot water. Sterilization of the milk is advocated in the case of all infants under 3 months of age for reasons presently to be discussed. After that time it may be discontinued, and pasteurization of the milk substituted until the eighth or ninth month, when raw milk may be used, provided the weather be cool, the milk reliable, and the use of the raw milk produces no digestive disturbance. During the summer it is better to pasteurize or to sterilize all milk used in infant feeding. All pasteurization and sterilization are, however, processes to be reserved for home use only. As a rule milk that has been commercially pasteurized or sterilized should not be used, as it may have been imperfectly kept by the dealer after the process.

Reasons for the sterilization and pasteurization of milk.—Apart from the safety the sterilization or the pasteurization of milk confers by virtue of the destruction of all its nonspore bearing bacteria (the word "sterilization" is not used here in the laboratory sense, but refers merely to measures which will destroy ordinary pathogenic organisms) there is abundant and incontrovertible evidence to show that by these measures both the morbidity and the mortality of infants from gastro-intestinal disease has been greatly reduced.

There are also additional and important reasons in the case of infants of less than 3 months of age which render the sterilization of the milk from their use especially desirable. Russell has shown that heating the milk destroys the tendency of the fat globules to coalesce and distributes them uniformly throughout the milk. This combined with the partial inhibition of the curding action of the gastric juices upon the casein of heated milk prevents the formation of large fat containing curds in the stomach.

Now, the gastric capacity of young infants is both absolutely and relatively very small. During the act of nursing, when the stomach has been filled a portion of its contents is passed on into the duodenum. That this must take place is readily shown by consulting the records of Feer's investigations and by comparing the amounts taken at single nursings with the absolute gastric capacity of infants of that age as determined by Pfaundler.

The soft, flocculent, diffluent curd of heated milk readily permits this action to occur as the stomach reaches the point of physiological distension.

Objections to the use of sterilized milk.—The use of sterilized milk for the feeding of infants has often been objected to, first, on account of supposedly greater difficulty in digestion, and second, because of the danger of producing infantile scurvy thereby. The first objection is founded upon misapprehension, as can readily be shown by comparing the action of rennet ferment on raw milk and on milk that has been previously heated. The raw milk coagulates firmly, while the heated milk has a soft, almost diffluent clot. Moreover, careful investigations of the digestive absorption of the constituents of heated milk have shown evidence of a considerably greater degree of completeness in such absoption than is the case in unheated milk. This, conjoined with the favorable experience of the French clinicians with heated milk, must be regarded as conclusive evidence of the superiority of both sterilized and pasteurized cow's milk present over raw cow's milk in this respect. The second objection, that of causing infantile scurvy, I believe, can be demonstrated to reside, in all probability, in qualities inherent in the milk used and not attributable to the mere fact of its sterilization. Scurvy has been seen not only as the result of pasteurized or sterilized milk, but also in breast-fed children and in those fed on raw cow's milk. We may, therefore, infer that certain constituents necessary to the nutrition of the body were not being supplied. At Professor Budin's clinic in Paris, at all the numerous milk depots ("Gouttes de Lait") scattered throughout France, where nothing but unsophisticated, sterilized cow's milk is used for the artificial feeding of infants from the earliest age, infantile scurvy is practically unknown. Budin tells us of a visiting physician who was unable to convince himself that sterilized milk did not produce scurvy. Budin invited him to inspect the infants who presented themselves with their mothers for their weekly inspections and weighings, as they are obliged to do. Every babe was stripped and the visitory was able to verify for himself that not one presented signs of scurvy or even of rickets in the slightest degree.

I would put forth tentatively the following development of Ralfe's theory as to the causation of scurvy as a possible explanation in this connection of the etiology of infantile scurvy and tending to remove this odium from whole, sterilized cow's milk, not too rich in fat. On theoretical grounds, scurvy may be regarded as a pathological condition caused by the diminution in the body of those alkaline bases which are necessary for the maintenance of a normal condition of health. These are ordinarily supplied in our food in the form of salts of the alkaline bases, especially potassium. Now, I would go a step farther and say that in order to undergo absorption during digestion, these salts must be supplied in combination with an acid radical which can be set free by the action of the digestive juices, such as phosphoric, citric, malic, and similar acid radicals. The negative proof of this contention is the rapidity with which scurvy is cured when the system is freely supplied with such salts.

I think two causes often going hand in hand are mainly responsible for the production of scurvy in infants. The first is an absolute insufficiency of the salts alluded to and the second is a relative insufficiency of these salts when compared with the fat present in the diet.

In regard to the latter condition we have seen how a diet excessive in fat may draw upon the alkaline bases of the body for the purposes of saponification. When they are being inadequately supplied in the food as well it is easy to see that the time would not be long in coming when the available supply would be depleted, radical changes wrought in the composition of the body tissues and the constitutional symptoms would follow.

In many of the cases of infantile scurvy caused by sterilized milk the formulæ used seems to have been the causative factor, i. e., low proteids or low proteids and high fat. Now all such modifications are derived from the dilution of top milks and creams with water. This implies that the quantity of the mineral salts present in the milk is greatly diminished, as, in order to produce this relative proportion of fat and proteid, small amounts of these top milks and creams are diluted with large volumes of water.

Thus a modified milk mixture of the following formula: Fat, 3; sugar, 6, and proteid, 1, is obtained by diluting 6 ounces of 10 per cent cream with 12 ounces of water and adding 1 ounce of milk sugar. This has at once the effect of reducing the mineral salts in this mixture to one-third the amount present in a similar amount of whole milk. When higher fat modifications derived from cow's milk are used without increasing the proteid, or when the proteid percentage is to be reduced, a richer cream must be taken in smaller amount and diluted with a greater volume of water. On the other hand, I would attribute the occurrence of cases of scurvy which have been observed to result from the use of whole sterilized cow's milk to the presence of an excessive amount of fat in the milk, which, by reason of the greater digestibility of sterilized milk, when compared to raw milk, was ingested without causing acute gastro-intestinal disturbance.

It is easy to see, then, how a milk modification in which the mineral salts are greatly reduced, or even a rich whole milk, which by virtue of its sterilization is thereby made easier of digestion, may, on the one hand either by deficient supplying of alkaline bases, and on the other by their excessive abstraction from the body for the purposes of saponification, produce in the long run the alteration of the body tissues and fluids which may result in scurvy. It may, however, be objected that the proprietary foods and condensed milk, which are anything but rich in fat, are themselves the most prolific causes of infantile scurvy.

This objection may be met by the fact that these are concentrated foods, and, for use, are diluted with large volumes of water. In the case of condensed milk, at least, this has the effect of reducing the salts far below the limit required by the body. Thus, condensed milk, when diluted with 6 parts of water, contains 0.17 per cent, with 12 parts 0.10 per cent, and with 18 parts 0.07 per cent of these salts. These are the dilutions ordinarily used in the feeding of infants. Taking woman's milk as a standard of infant needs in this respect in maternal nursing, at least, we find, according to Von Bunge, that potassium and sodium are by far the preponderating alkaline bases in its salts, and that the child requires of them 0.07 and 0.025 per cent, respectively, in its food.

This, however, may be said to be true only when lactation is well established. During the first weeks of lactation the percentage of mineral salts present is higher than this, which may have the effect of increasing the reserve supply. As these salts are present in whole cow's milk in the proportions of 0.17 and 0.05 per cent, it will be seen that the dilution of condensed milk as given above reduces them to infinitesimal amounts, in the case of the first dilution 0.00309 and 0.00085 of each. Nor does it necessarily follow that an amount of these salts similar to that furnished with human milk will be adequate when supplied in conjunction with other foods, as much depends upon the conditions governing absorption in the alimentary tract. In the case of the proprietary foods, scurvy has been met with in those cases where they have been used as a complete substitute for milk. In some of these foods, such as Nestle's, Eskay's, Ridge's food, and Imperial Granum, the amount of inorganic salts present, differing but little or being much less than those in condensed milk when diluted to the extent required for use, predicates a similar condition on their part. When we come to consider artificial foods in general I think we are justified in assuming that they should contain an amount of inorganic salts at least equal to that of the food, i. e., cow's milk, which has been the most successful in the artificial feeding of infants. When we reduce cow's milk to the basis of the relative proportion of its solid constituents to each other we find that cow's milk has the following average composition:

	Per cent.
Fat	31.25
Sugar	35.16
Proteid	27.34
Salts	6.25
	100.00

There seems to be no proprietary food on the market that approaches cow's milk in the respect of its content of inorganic salts in proportion to its other ingredients, the nearest being in the case of a food (Garnrick's) which contains 1.42 per cent of inorganic salts, and of which considerably over one-half of its content of carbohydrate is insoluble, a condition which must certainly be taken into account when considering the availability of such salts for absorption.

We may, therefore, with reason, I think, dismiss our fears of the production of infantile scurvy by the use of sterilized or of pasteurized cow's milk, administered in suitable quantities, provided its inorganic salts are not reduced too greatly by dilution nor its fat content excessive (over 3 per cent). If these conditions are complied with, I am convinced that the dangers of scurvy from its use have been greatly overestimated. At all events, the danger involved from the use of sterilized or of pasteurized milk with respect to scurvy is so small, under these conditions, in comparison with the advantages to be derived that they may be disregarded. As an additional prophylactic when desired, however, we may avert all danger by the administration once or twice a day of a small quantity of orange juice (say 15 to 30 cubic centimeters) one hour or so before feeding. It is needless to say that the juice so administered must be from perfectly fresh fruit and strained free from particles of skin.

PASTEURIZATION OF MILK.

This consists in the heating of milk to 60° C. (140° F.) for twenty minutes. While insufficient for the complete sterilization of milk, it destroys most of the nonspore-forming micro-organisms, including the pathogenic germs, besides not altering materially the taste of the milk. It is therefore recommended for milk to be used in feeding infants from the third month on, at least during the summer months.

METHOD OF PASTEURIZING MILK.

Water is brought to a boiling point in some utensil with a close-fitting lid. The utensil is then removed from the stove and placed on some nonconductor of heat, as a square of asbestos or a board. The feeding bottles are stood up to the level of the milk in them in the water, the utensil covered, and the whole left for twenty minutes. The milk bottles are then rapidly cooled by the use of cold water on the exterior of the bottles and are kept on ice until required.

It is highly important to remember that neither sterilization nor pasteurization will make bad or stale milk good, and that once sterilized or pasteurized it requires the same care in preservation as raw milk. Very convenient forms of apparatus, such as Arnold's or Soxhlet's, for sterilization, or Freeman's for pasteurization of milk can be bought at the shops.

ARTIFICIAL FEEDING OF INFANTS UNDER ONE MONTH OF AGE.

When we consider the composition of woman's milk in the early period of lactation, we are impressed with the fact that while the proteids are high, the sugar and fat are lower than at subsequent times. This has the effect of reducing its caloric value, and is doubtless dependent upon the needs of the infant in this respect.

We can, therefore, more easily imitate the provisions of nature by feeding skimmed milk to infants in the first month of life. Walls has found[a] that sterilized undiluted skimmed milk is entirely digestible even by premature infants. As the energy quotient required by infants does not become high until the second week, it may be assumed that skimmed milk will more nearly meet their requirements at this age than whole cow's milk.

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[a] F. X. Walls, Jour. Am. Med. Assn., 1907, Vol. XLVIII, pp. 1389-1392.

Skimmed milk is obtained either as centrifugally skimmed milk from the dairy or by siphoning off the under half of a quart bottle of milk whose cream has risen.

Skimmed milk has the following average composition:

	Per cent.
Fat	0.50 to 1.00
Proteid	3.50
Sugar	4.50
Salts	0.75
Water	90.75 to 90.25

Caloric value per kilogram, 374.5 to 421.

The amounts to be taken are determined as previously explained. After the end of the first week one third whole milk and two-thirds skimmed milk can be given; after the end of the second, one-half whole milk and one half skimmed milk; at the end of the third week, three-quarters whole milk and one-quarter skimmed milk, passing to whole milk at the beginning of the first month.

FEEDING OF OLDER INFANTS.

The seventh month of infancy marks the time when it is desirable to supplement exclusive milk feeding by some other food. This should take the form of some cereal broth, and should be added to the milk in the proportion of one-third broth to two-thirds milk.

The preparations recommended for this purpose are dextrinized gruels, oatmeal jelly, and barley water.

Dextrinized gruel. Make a thin paste of cold water and 1 or 2 heaping tablespoonfuls of barley, wheat or rice flour, add 1 quart of boiling water, and boil for fifteen minutes in a double boiler. When the gruel is cool enough to be easily tasted, dextrinize by adding 1 teaspoonful of diastase solution. An active solution of diastase may be prepared by soaking a tablespoonful of crushed malted barley grains in sufficient cold water to cover them (about 2 tablespoonfuls) and placing the mixture in the refrigerator over night. In the morning the liquid which resembles weak tea is strained off. A tablespoonful of this fluid will dextrinize a pint of gruel in fifteen minutes. Or, a good commercial preparation of diastase may be used.

Oatmeal jelly. To 2 tablespoonfuls of oatmeal add 1 quart of water and boil for three hours, keeping up the quantity to 1 quart by the addition of water as it boils away. Strain through coarse muslin. As this forms a jelly when cold, it should be added warm to the food.

Barley water.—This is prepared in the same way. Barley grains or barley flour may be used. If the former, soak the grains in water over night.

METHOD OF INCREASING THE INFANT'S DIET.

As long as an infant is making satisfactory gains no change in the daily quantity of food is required. To this end, all artificially fed infants should be carefully weighed each week and the weights noted for future reference. When the rate of gain for a week has suffered diminution, in the absence of other symptoms to account for it, we are to know that the time has come for an increase in the diet. The amount of this increase is determined along lines previously laid down, i. e., by weighing the infant and giving it that proportion of food to its body weight indicated by its age.

We should be watchful for symptoms of overfeeding with every increase instituted in the quantity of the daily food. As long as the stools are normal in number, color, quantity, and consistency, and the urine remains limpid, no fear of overfeeding may be entertained.

When, however, loss of appetite is manifest, the bowels are constipated, pale, formed, and dry, the infant is being overfed, and a reversion to the former amount of its diet, or the substitution of skimmed milk for a few days must be employed until these symptoms disappear.

PRECAUTIONS TO BE OBSERVED IN THE ARTIFICIAL FEEDING OF INFANTS.

Every utensil used in the preparation of infant food should be clean. This does not mean a mere macroscopical cleanliness, but surgical cleanliness as well. Vessels used to hold the infant's food during its preparation should be scalded with boiling water after previous thorough cleansing. Feeding bottles are to be cleaned after use, first with cold water, and then with warm water and some alkaline soap powder. Adhering particles of milk are to be removed with a bottle brush. The bottles are to be sterilized by boiling them in water, and storing them in an inverted position, when empty, to prevent the access of dust to their interior. When new nursing bottles are bought, in order to prevent them from cracking from the extremes of cold and heat to which they are subject, they should be annealed. This is accomplished by placing them in cold water, bringing the water to a boil, and allowing the bottles to remain in the water until it is cold.

Only rubber nipples fitting on the necks of the bottles should be used. One should be able to turn them inside out for cleansing purposes. The hole in the top should be just large enough to allow the milk to drop rather rapidly when the bottle is inverted. If it issues in a stream the hole is too large. Nipples before use should be boiled, and may be kept in a saturated solution of boric acid. In feeding the child care should be taken to hold it in such a position that it can easily take its food. A child should not be coaxed to take more food than it desires at the time, and its wishes in this matter should be treated with respect. Any portion of food left after a feeding should be thrown away, and on no account should it be used again.

While, as a rule, it may be postulated that no infant is born with a digestion congenitally weak, still, as the result of inadequate feeding, both maternal and artificial, we do encounter infants whose digestive processes are a law unto themselves. The efficient nutrition of such infants often presents a problem which must be attacked upon individual lines. The investigations of Teixeira de Mattos,[a] Salge,[b] and others have shown that fat-free buttermilk, or equal parts of buttermilk and malted cereal broths, are in many instances digestible with apparent satisfaction by such infants. As skimmed milk, also, is closely related to buttermilk in its composition, its use as an article of diet (sterilized) under these circumstances is warmly recommended. As soon as tolerance for cow's milk in this form is established, it must, however, be supplanted by a gradual return to whole cow's milk, as both buttermilk and skimmed milk are too poor in nutritve elements to furnish the basis for any long-continued scheme of artificial feeding.

It should not be forgotten that atrophic infants require a greater energy quotient than the normal child of the same weight. This is due to two reasons, first, by reason of the greater radiation of heat on account of their deficiency in bodily fat, and second, because their proportion of living body cells is greater in respect to their weight than is the case in infants of normal nutrition. In the latter, 8 to 12 per cent of their weight consists of fat, whose function in the metabolic processes of the organism consists only in furnishing a storehouse for energy and in conserving the bodily heat. In the emaciated child of the same weight, the body consists almost entirely of cells performing vital functions, all of which require nutriment for their proper performance. In view of these facts, in such cases the food given may be increased above the normal both in quantity and in caloric value, taking care, however, not to provide such an excess that the digestion is thereby embarrassed, and to reduce the nourishment to amounts appropriate to the weight and age of the child as the normal average of weight for age is approached.

It is also important to remember that cow's milk when compared with human milk is essentially an alien food. Both its fats and its proteids are different in composition from those of human milk and, being adapted to the nourishment of an animal on a different zoological plane, must of necessity be regarded as substances foreign to the human infantile digestive tract. As a consequence, greater energy is required for its digestion and assimilation, and it is of the highest importance that the infant metabolism be not further strained in this connection by the imposition upon it, in addition to this task, of the conversion of a milk whose digestibility is further impaired by fermentative changes due to its improper preparation and preservation as a food.

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[a] Teixeira de Mattos. Die Buttermilch als Säuglingsnahrung. Jahrbuch f. Kinderheilk., 1902, pp. 1-61.

[b] B. Salge. Buttermilch as Säuglinsnahrung. Jahrb. f. Kinderheilkunde. 1902, 157-161.

While we can never hope to vie with natural nursing, an application of the principles briefly expounded in this paper will go far, I am convinced, toward eliminating the excessive complexity and uncertainty which have hitherto characterized the whole subject of infant feeding, and, in the main, be productive of better results than we can obtain by other methods.

24. THE RELATIVE PROPORTION OF BACTERIA IN TOP MILK (CREAM LAYER) AND BOTTOM MILK (SKIM MILK), AND ITS BEARING ON INFANT FEEDING.

THE RELATIVE PROPORTION OF BACTERIA IN TOP MILK (CREAM LAYER) AND BOTTOM MILK (SKIM MILK), AND ITS BEARING ON INFANT FEEDING.[a]

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In the course of a study of tubercle bacilli in market milk[b] and of a later study of the best procedure for their detection in milk it was noticed that when guinea pigs were inoculated with the cream a very much higher percentage died from acute infections than when the sediment was used. The inference was natural that the cream contained more bacteria than the bottom milk or sediment. A few preliminary examinations having shown this supposition to be correct, a study was begun as to the number of bacteria in the whole milk, the bottom milk or sediment, and the cream, both that collected by gravity and by centrifugation.

The relative number of bacteria in the top milk and in the bottom milk is a subject of very great importance in the modification of milk for infant feeding. All the writers on pediatrics and infant feeding give formulæ for the modification of milk based upon the use of various amounts of top milk. My studies show that top milk, such as is advised for use in the above formulæ, contains from 10 to 500 times as many bacteria per cubic centimeter as the mixed milk. This preponderance of bacteria in top milk may account for the fact that sometimes children do not thrive on modified milk when made from top milk, but improve when the whole milk is used for modification.

The various bacteria causing acute infections, as well as tubercle bacilli, are more numerous in the top milk than in the bottom milk. In many cases this difference is more than a hundredfold and, as infection must depend to some extent on the number of bacteria intro duced into the body, too little attention has been given to the question of the number of bacteria in top milk when used for infant feeding. Oftimes when infants are taken off breast milk and put on modified cow's milk made from top milk it is found that, in spite of various modifications containing varying percentages of proteid, the milk fails to agree with the infant. In those cases which develop diarrhea the fault may not be in the proteid, but in the large number of bacteria in the top milk used for the preparation of the formula.

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[a] This is a summary of a paper read before the American Public Health Association in Winnipeg, Canada, August 31, 1908.

[b] Anderson, John F.: "The frequency of tubercle bacilli in the market milk of the city of Washington, D. C." Bul. No. 41, Hyg. Lab., U. S. Pub. Health and Mar. Hosp. Serv., Wash., 1908, p. 163.

The greater frequency of intestinal tuberculosis in young children may be due, not alone to the fact that they use a large amount of milk, but because top milk, which contains more tubercle bacilli per cubic centimeter than the whole milk, is used in the preparation of modified milk formulæ.

The literature upon the subject of the relative number of bacteria in top milk and in bottom milk is very slight. None of the writers seem to have realized the great importance of the subject in its relation to infant feeding.

In 30 samples of milk examined the average number of bacteria in gravity-raised cream was 69,211,000 and in the sediment layer 4,360,000 bacteria per cubic centimeter.

In 26 samples of milk the average number of bacteria in gravity and centrifugally raised cream in the sediment and in the mixed milk was—

Gravity.		Centrifugalized		Whole milk.
Cream layer.	Sediment layer.	Cream layer.	Sediment layer.	
[illegible]	4,840,000	26,840,000	18,840,000	14,388,000

In 6 samples of milk the average relative number of bacteria in the gravity cream was 15,416,000; skim milk, 2,050,000; in the sediment layer, 1,405,000; and in the whole milk, 2,708,000.

In 7 samples of milk the average relative number of bacteria in the centrifugally-raised cream was 4,500,000; in the sediment layer, 725,900; in the skim milk, 119,700; and in the whole milk, 619,800.

One sample of milk contained 500 times as many bacteria per cubic centimeter in the cream as in the bottom milk.

When milk is centrifugalized the great mass of bacteria go up with the cream; a lesser number is carried down in the sediment. The skim milk contains many times fewer bacteria per cubic centimeter than the cream or sediment layers.

Centrifugally raised cream contains more bacteria per cubic centimeter than the gravity-raised cream from the same milk.

25. NATIONAL INSPECTION OF MILK.

NATIONAL INSPECTION OF MILK.

By Harvey W. Wiley, M. D., Ph. D.,
Chief Bureau of Chemistry, Department of Agriculture.

Much can be done in improving the character of milk by inspection under the national law of June 30, 1906. This law applies only to milk sold in the District of Columbia and the Territories and the milk entering into interstate commerce. There are, however, many large cities located on or near the boundaries of States, and in these cities a large part of the milk supply comes from without the State in which they are situated. New York, Philadelphia, Cincinnati, Louisville, Chicago, St. Louis, and Kansas City are types of cities of this class. Under the provisions of the law steps have been taken to determine the character of the milk supply furnished some of these cities, in so far as its chemical composition is concerned and incidentally the inspection of dairies has necessarily been made. In all, about 3,500 samples of milk have been secured and examined. The number of producers, however, was not so great, as often a great number of samples were obtained from one producer. The manner of conducting the milking and the shipping of milk into various cities was studied and in all cases where samples were collected for prosecution the inspectors accompanied the shipments, riding in the milk cars so as to be certain to maintain the identity of the samples and to be able to swear to their genuineness after they had crossed the State lines. The samples were at once taken to the local laboratories of the Bureau of Chemistry, in the cities mentioned, and subjected to a preliminary examination, and those which showed adulteration or misbranding were afterwards submitted to a more careful examination in order to establish with certainty a case sufficiently strong to warrant prosecution. It was deemed wise to prosecute only the flagrant cases, but many samples were found by analysis to be watered or partly skimmed, and these were not prosecuted because of the difficulty which it was thought would arise in proving conclusively to the jury that the milk had been tampered with.

Owing to the necessary delay in the prosecution of cases in the Federal courts only 57 cases so far have gone to trial. Of these, 53 entered a plea of guilty of watering or skimming or both, 1 of adding a preservative, 1 of watering and adding a preservative, 1 was convicted of adding water, and another charged with the same offense was acquitted by the jury. A curious incident is connected with this, in that the case in which the jury acquitted the defendant was the most flagrant, while the case following was a much less flagrant case of watering.

In the case in which the verdict was rendered for acquittal the milk was grossly adulterated and practically no evidence was submitted by the defendant. The evidence submitted by the Government was strong and well connected and proved without a shadow of a doubt the adulteration charged, namely, both watering and skimming. The chemical evidence in this connection was exceptionally strong and convincing. Sixteen additional flagrant cases have been reported to the district attorney in Illinois regarding samples shipped from Illinois into St. Louis. In 2 cases it was found by analysis that the milk was skimmed and in 14 watered. Twenty-six cases have been reported to the district attorney in the cities adjoining Chicago for prosecution for adulteration and misbranding milk sent into that city; 12 of these were found to be skimmed, and 14 watered. Sixty-two cases have been reported to the district attorney in Kentucky and Indiana adjoining Cincinnati, and 9 cases in Kansas City. It is evident that if a jury is made up of those who produce, it is hard to get a conviction. In addition to the 113 cases which are of the strongest possible character on which conviction could be easily secured a much larger number is evidently adulterated by skimming or misbranding, but these were not reported for trial. When very rich milk is watered or partly skimmed and the resulting fat content is still well above the standard, only very strong evidence is likely to convict. The general result has shown that a large percentage of the milk going into some of our great cities is either watered or skimmed, or both. It is very gratifying, however, to find from the investigations that almost no milk samples have been treated with preservatives.

It is evident from the above brief summary that the national law is to be a great help to the state and municipal authorities in controlling adulterations and misbranding of milk.

26. THE MUNICIPAL REGULATION OF THE MILK SUPPLY OF THE DISTRICT OF COLUMBIA.

THE MUNICIPAL REGULATION OF THE MILK SUPPLY OF THE DISTRICT OF COLUMBIA.[a]

By Wm. Creighton Woodward, M. D., LL. M.,
Health Officer of the District of Columbia.

I. THE DEVELOPMENT OF THE MILK-INSPECTION SERVICE.

Milk is a food. Legislation for the regulation of the milk supply is enacted with that fact in mind, and not infrequently legislation relating to the manufacture and sale of foods generally is applicable as a whole or in part to the production and sale of milk. For these reasons it has been deemed expedient in discussing the municipal regulation of the milk supply to refer rather oftener to the regulation of the general food supply than otherwise would have been necessary. It is interesting, too, to note how changes in our modes of living, or increasing knowledge with respect to the sanitary and mercantile relations of foodstuffs, have altered our practice with reference to governmental supervision and control. The inspection of flour, of salted provisions, of tobacco, and of spirituous liquors, in the cities of Washington and Georgetown, in the District of Columbia, was early provided for, but the regulation of the sale of fresh meats and of milk is of comparatively recent origin.

The first legislation relating to the sale of milk of which I have been able to find record was enacted by the board of aldermen and board of common council of the city of Washington on August 1, 1863. The ordinance was entitled, "An act in relation to cows," and seems to have been directed rather against the nuisance liable to arise from the keeping of cows than against any supposed effect which insanitary conditions in and about cow yards, pens, and stables might have upon the milk issuing therefrom.[a] Apparently, however, it was construed to prevent the selling of milk under certain conditions since we find enacted by the sixty third council, on May 24, 1866, an act entitled, "An act explanatory of the act entitled, 'An act in relation to cows,' approved August 1, 1863," which provided simply that the first section of the earlier act should be so construed as to permit the selling of milk by persons who keep one or two cows.[b] The situation in Georgetown, then a separate corporation, must have been similar to that existing in Washington, for on April 22, 1865, the board of aldermen and board of common council of the corporation of Georgetown enacted an ordinance substantially the same as that previously enacted by the corporation of Washington, but further provided that no person should be permitted to feed or milk a cow on any of the public streets or footways of the city.[c]

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[a] For the information of readers not familiar with the administration of municipal affairs in the District of Columbia, a memorandum descriptive thereof is appended. (See page 789.)

The next record of action looking toward the regulation of the food supply of the District of Columbia appears in the act of Congress, entitled "An act to provide a government for the District of Columbia," approved February 21, 1871, which, as an incident to the general reconstruction of the local government, created a board of health and in terms made it the duty of that board to prevent the sale of unwholesome food in the cities of Washington and Georgetown.[d] This board seems to have proceeded with admirable promptness and energy to the discharge of its duties, for on May 15, 1871, within about five weeks after the day of its organization, the board took steps to prevent the sale of unwholesome food in the cities of Washington and Georgetown by enacting an ordinance for that purpose. This ordinance in so far as it relates to the sale of milk was as follows:

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[a] An act in relation to cows.

Be it enacted by the board of aldermen and board of common council of the city of Washington, That from and after the first day of October, eighteen hundred and sixty three, it shall not be lawful for any person or persons to keep, provide for, or maintain within the limits of the city of Washington, a cow yard, pen, or stable for dairy or other purposes, nearer than two hundred feet of any dwelling house, other than the dwelling house of the owner or keeper of such yard, pen, or stable, under a penalty of not less than one nor more than five dollars for each day's offense so continued; to be prosecuted and recovered as other fines and penalties due the corporation are prosecuted and recovered: *Provided, however*, that nothing herein contained shall apply to persons who keep but two cows for their own immediate use.

SEC. 2. *And be it further enacted*, That the owner or keeper of any cow yard, pen, or stable, or other place where cows are kept, within the limits of the city of Washington, shall daily remove the filth from and keep clean such yard, pen, stable, or other place, under a penalty of not less than one or more than five dollars for each and every offense; to be recovered as provided for in the first section of this bill.

The commissioners of the several wards and police officers of the city are instructed to report and prosecute any and every infringement of this act.

Approved August 1, 1863.—(Laws of the corporation of the city of Washington passed by the sixty-first council, chap. 4.)

[b] Laws of the corporation of the city of Washington, passed by the sixty-third council, chap. 27.

[c] Ordinances and resolutions of the corporation of Georgetown, 1865, page 24.

[d] 16 Stat. L., 424.

> SEC. 2. *And be it further ordained and enacted,* That no person shall manufacture, prepare, or sell any liquor used for drink, whether malt, vinous, or ardent, or milk of cows or goats, intended to be used as food or drink, which has been adulterated with any poisonous or deleterious ingredient; and any person violating the provisions of this section shall, upon conviction, be punished by a fine of not less than fifty nor more than five hundred dollars for each and every such offense.
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> * * * * * * *
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> SEC. 7. *And be it further ordained and enacted,* That no person shall offer for sale, or keep for such purpose, any unwholesome, watered, or adulterated milk, or swill milk, or milk from cows kept up and fed on garbage swill or other deleterious substance; nor shall any person make for sale any butter or cheese from such unwholesome milk; and any person violating the provisions of this section shall, upon conviction, be punished by a fine of not less than five nor more than twenty-five dollars for each and every such offense. Passed May 15, 1871.[a]

Strange as it may seem to the sanitarian of to-day, and yet apparently quite in keeping with the then prevailing ideas, while this ordinance very clearly and positively required that places where meat and vegetables were sold for food should be kept in a clean and wholesome condition, and that meat and vegetables should not be allowed to become poisoned or infected or unfit for food, no such provision was enacted with respect to the sale of milk. The board of health held, however, advanced ideas with respect to the production of milk for sale and in its first annual report says:

> The proper diet of cows is also a measure of vital moment to that large class of infants and others who subsist chiefly on milk and its preparations. The deterioration of this most nourishing secretion by swills and other nefarious compounds has, in our large cities, vastly increased the percentage of deaths from diarrhea and cholera infantum.[b]

In the second annual report of the board of health, under date of October, 1873, the food inspectors in the service of the board, Messrs. William Wolf and Robert Wilson, and Dr. Charles Allen, drew attention to the importance of a good milk supply. These inspectors seem to have realized even then the importance of the inspection of milk at the place of production, a feature of milk inspection service that on the part of sanitarians generally did not receive the consideration that it deserved until about twenty years later. Their practical experience must have taught them, too, the only way in which the milk supply of the District could ever be controlled at its source, viz, by a system of permits or licenses, a method which was adopted in the District in 1895 and which has since come into more or less general use throughout the country. What these men say with respect to this matter, in view of the time when it was said, is well worth quotation at length:

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[a] Report of Board of Health, 1872, pp. 63, 64.

[b] Report of Board of Health, 1872, p. 18.

> The milk supply of this District is from dairies established in the county, and in the neighboring States of Virginia and Maryland; and as a proper inspection would include the examination and sanitary control to some extent of the dairies, as well as the milk when offered for sale here, we recommend that the board require dealers to procure permits before they can dispose of their milk here. By this means supervision might be obtained over them, even in Virginia and Maryland, and we doubt not that the dairymen would readily adopt suggestions looking to the proper preservation of their milk from unhealthy contaminations.[a]

And the then health officer, Dr. P. T. Keene, in submitting to the board of health the report of the food inspectors, recommended that "the regular inspection of dairies and the requirement of licenses to vendors of milk under strict provisions should at once be instituted."[b]

In its third annual report, covering the year ending September 30, 1874, the board of health published a statement by Dr. B. F. Craig, its chemist, who had analyzed a number of samples of milk, most of which, said Doctor Craig, "appeared to have some portion of the cream removed, or else to have been originally of poor quality." After commenting upon possible sources of error in the use of the lactometer and upon the importance of chemical examination, he adds:

> but before chemical examination can produce any effect there should be a legal definition of the character of what can be sold as milk and what may be sold as skimmed milk, and, I would suggest, of what may be sold as cream. The law can at present take hold of nothing but the proved addition of water or other adulterants. It should also be made to cover the case of removal of cream, and in fact to exclude from the market all milk below a certain quality, that quality being determined by the amount of water in the milk and the amount of fatty matter or cream to be obtained from it.[c]

The health officer, in his report to the board of health for 1875, again calls attention to the importance of inspecting milk at the places of production:

> For meat and milk, per example, the most important of all, we are entirely at the mercy of the producers, and must continue so to be until the abattoir, or some other system, be established by which it may be possible to inspect every animal at the time of slaughtering, and until some carefully organized plan of checking adulteration of milk be inaugurated. * * * And this I find

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[a] Report of Board of Health, 1872, p. 124.

[b] Report of Board of Health, 1872, p. 121.

[c] Report of Board of Health, 1874, pp. 206, 207.

is the principal acted upon in many of the European cities. Aiming to control the fountains of supply, the authorities largely prevent deleterious and adulterated food from reaching the hands of the retailers.[a]

The board of health, in its annual report for 1877, the last report it was ever to issue, again called attention, but only in a general way, to the importance of supervising the sale of milk, and particularly to the relation between the production of milk under insanitary conditions and the unwholesomeness of the article produced.[b]

For several years past the board of health had had the services of an analytical chemist to assist it in procuring and maintaining the purity and wholesomeness of the food supply, but with the advent of the fiscal year 1876-77 his name disappears from the records. The situation is graphically shown in a resolution that appears in the minutes of the board for June 30, 1876:

> On motion of Professor Langston it was ordered that all employees of the board except the poundmaster and the force serving under him be discharged to take effect this day.

The board of health was being strangled to death by the withdrawal of the funds necessary for its operations. Arrangements were made for the continuance of the work of the board with a very much reduced force, but the board died on June 11, 1878. No provision was ever again made for the appointment of an analyst for the board or for its successor, the health department, until July 28, 1892. In the meantime, in so far as related to the analysis of foods and other articles, the board of health and the health officer had to rely on such outside assistance as they might be able to obtain. Although it had been charged, by law, with the duty of preventing the sale of unwholesome food in the cities of Washington and Georgetown, and endowed with broad legislative power, no action seems ever to have been taken by the board to regulate the milk supply of these cities further than to promulgate the ordinance of May 15, 1871,[c] and to enforce that ordinance in such manner as its available force would permit. There had been much in the way of suggestion and recommendation for improvement, but nothing in the way of action. The time was not yet ripe.

The board of health of the District of Columbia became extinct with the passage of an act entitled "An act providing a permanent form of government for the District of Columbia," approved June 11, 1878, and its legislative power died with it. The new law provided, with respect to the board of health, as follows:

> That in lieu of the board of health now authorized by law, the Commissioners of the District of Columbia shall appoint a physician as health officer, whose

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[a] Report of Board of Health, 1875, p. 72.

[b] Report of Board of Health, 1877, p. 33.

[c] See page 749.

duty it shall be, under the direction of the said Commissioners, to execute and enforce all laws and regulations relating to the public health and vital statistics, and to perform all such duties as may be assigned to him by said Commissioners; and the board of health now existing shall, from the date of the appointment of said health officer, be abolished.[a]

Although the act itself set forth that the health officer should be appointed in lieu of the board of health, as a matter of fact his duties were substantially the same as those of the health officer, who had previously operated under the direction of the board of health; the Commissioners of the District were in fact, if not in law, the successors to the board of health. Neither the Commissioners nor the health officer, however, were authorized to promulgate regulations relating to public health, but were authorized merely to operate under such laws and regulations relating to such matters as were then in force or might thereafter be enacted by Congress. And Congress, having assumed by the act of June 11, 1878, exclusive legislative control of the food supply of the District, made its first move toward that end on January 25, 1879, by passing an act entitled "An act for the protection of dairymen, and to prevent deception in sales of butter and cheese in the District of Columbia." The act, as its title imports, was solely for the protection of dairymen against unfair competition resulting from the fraudulent sale of oleomargarine in the District of Columbia and for the protection of the community from such sales. It had no relation whatsoever to matters of health, and frankly permitted the sale of oleomargarine when properly marked.

The validity of the ordinances of the defunct board of health appears to have been soon questioned, and on April 24, 1880, Congress, by a joint resolution entitled, "Joint resolution legalizing the health ordinances and regulations for the District of Columbia," legalized certain of the ordinances enacted by the board of health, among them an ordinance to prevent the sale of unwholesome food in the cities of Washington and Georgetown." These ordinances and the ordinances relating to the location and keeping of cow yards, pens, and stables, previously enacted by the boards of aldermen and common councils of the corporations of Washington and Georgetown, represented at this time the entire body of law in force in the District of Columbia relating to the production and sale of milk. Crude as it appears, it was probably in keeping with the then prevailing ideas concerning the regulation of the production and sale of this food, although it was very far behind the needs of the situation, as viewed by the board of health itself and as set forth in its several annual reports.

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[a] 20 Stat. L., 107.

[b] 20 Stat. L., 264.

[c] 21 Stat. L., 304.

All questions relating to the production and sale of milk seem to have been in abeyance, and the dairy farmer and the milk dealer, the sanitary authorities, and the consumer of milk rested content with existing conditions until the fiscal year 1882-83. We find then, in the report of the health officer, Dr. Smith Townshend, for 1883, the following suggestion, contained in a report by Dr. B. G. Pool, medical sanitary inspector. The statement refers to certain investigations that Doctor Pool had made to ascertain the causes of cases of diphtheria, scarlet fever, and typhoid fever:

> On inquiry as to the source of milk supply, it was found that many persons were unable to give the name or residence of their milkman, seeming to consider themselves fortunate if they were able to secure the service of a "countryman." It is suggested as desirable that some provision should be made for the regular inspection of the sources of milk supply, not only as to the quality of the milk itself, but that inquiries be made to ascertain the prevalence of contagious diseases among the families of the milkmen.[a]

Although no record appears of any effort having been made to procure the enactment of the legislation necessary for the establishment and maintenance of a system of milk inspection embodying the supervision and control of places of production and sale, yet the health officer undertook in the following year to inspect the dairy farms from which the milk supply of the community was drawn. In his report for 1883–84, after recounting certain facts tending to show the importance of the proper supervision of the milk supply, he says:

> With such facts as these before us it becomes apparent that in making an examination to ascertain as to the comparative purity or impurity of the milk supply of a city the health officer must go farther than the making of an analysis of samples of the various milks sold. His influence must be felt by the producer as well as by the middleman who comes between the producer and the consumer.

The entire subject is discussed in a thoroughly scientific spirit, but the report does not set forth with any satisfactory detail the results of the investigation which was made, nor does it appear that any action was taken even at this time looking toward the establishment of a proper milk-inspection service.[b] The health officer, like his forerunner, the board of health, was moving in advance of the times.

Current reports in the spring of 1888 seem to have alleged the prevalence of adulteration of food and drink in the District, for on April 10, 1888, the health officer calls attention of the Commissioners to the fact that the health department is without an analyst, and in his annual report for that year he states that the inspector of asphalts and cements of the engineer department, who has been analyzing for the health department certain samples submitted to him, had very much reduced, and would probably further reduce, the amount of work which he performed for the health department. The appointment of a chemist for the health department was recommended.[a] On the 12th of October, of the same year, the first serious effort to regulate the food supply of the District of Columbia that had ever been made consummated in the enactment by Congress of an act entitled, "An act to prevent the manufacture or sale of adulterated food or drugs in the District of Columbia."[b]

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[a] Report of the Health Officer, 1882–83, p. 39.

[b] Report of the Health Officer, 1883–84, pp. 15 et seq.

The food and drugs act of October 12, 1888, was broad in its character, seeking to prevent within the District of Columbia the adulteration of foods and drugs generally. The term "food," as used in the act, was defined, however, to include every article used for food or drink by man, other than drugs or water, and therefore included milk. The general direction and control of the enforcement of the act were entrusted to the Commissioner of Internal Revenue; it was specified that the analysis provided for in the act should be under the control of the Commissioner of Internal Revenue, under such rules and regulations as might be prescribed by the Secretary of the Treasury, and the Commissioner of Internal Revenue was vested with authority even to declare certain articles or preparations to be exempt from the provisions of the act. By virtue of authority conferred by this act, the Secretary of the Treasury, under date of November 20, 1888, promulgated certain regulations concerning analysis of foods and drugs in the District of Columbia. He undertook to fix standards for certain specified foods, among them milk, and specified certain substances as "known to be injurious to health when present in foods." Others he described as "known to produce more or less toxic effects, and whose use in food is therefore harmful." Other substances were designated as "harmless coloring matters." The standard fixed for milk was as follows:

> Milk: Whole (pure) milk, the minimum specific gravity, "actual density," shall be 1.030 at 60° F., and the milk shall contain not less than 13 parts in 100 of solids, as follows: Fat, 3.5; solids, not fat, 9.5; water, not more than 87. The removal of cream, the addition of water, foreign fats, or coloring matter will be considered adulterations.

A form was provided upon which any person entitled under the law to have a sample of any food or drug analyzed might make application to the Commissioner of Internal Revenue for that purpose. It was required that applications be made in triplicate, one to be returned to the applicant with the report of the analyst, another to be filed with the United States district attorney, and the third to be retained by the Commissioner of Internal Revenue.[c]

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[a] Report of the Health Officer, 1887-88, pp. 24, 25.

[b] 25 Stat. L., 549.

[c] Instructions to Internal Revenue Officers, Series 7, No. 15, Nov. 20, 1888.

The means had been provided by which any interested person, whether a mere private purchaser or a representative of the health department, could procure an analysis, the ordinary purchaser under any circumstances, but the representative of the health department only when he suspected that the sale had been made in violation of law. Further than this nothing was done. The Commissioner of Internal Revenue waited for the health officer. The health officer waited for the Commissioner of Internal Revenue. The result was inevitable.

In his report for 1889, the Commissioner of Internal Revenue writes:

> No samples were submitted to me for analysis as provided by that act (act of October 12, 1888). This office is of the opinion that the failure to forward samples of suspected food for analysis may be ascribed to the apathy of the general public and that of the health department of the District of Columbia.[a]

One sample was received in 1891, but the nature or origin of it is not set forth. Two samples of milk were analyzed in 1892, but as to the origin the report is silent.[b]

The act making appropriations for the expenses of the District of Columbia, approved July 14, 1892, authorized the appointment of "one sanitary and food inspector, who shall also inspect dairy products and shall be a practical chemist." On July 28, of the same year, John D. Hird was appointed and entered upon the discharge of his duties. The makeshift for a chemical laboratory that was then provided was not ready for use until December, but no effort was made even during the interval to operate under the food and drugs act of October 12, 1888.[c] After the laboratory was ready for use successful prosecutions were brought under District ordinances, for the sale of colored milk and of milk that had been watered, but it was found that these ordinances provided no penalty for the sale of skimmed milk. Recourse was thereupon had to the act of Congress of October 12, 1888, relating to the manufacture and sale of adulterated foods and drugs in the District of Columbia, and to facilitate the operations of the health officer, the Secretary of the Treasury designated the chemist of the health department as an analyst to make analyses under the provisions of that act.[d] The Commissioner of Internal Revenue, in his report for 1894, writes:

> The act of October 12, 1888, to prevent the manufacture or sale of adulterated foods or drugs in the District of Columbia, imposes upon this office the duty of analyzing all samples submitted for decision as to their character. No provision has been made, since the first year of its passage, for increasing

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[a] Report of Commissioner of Internal Revenue, 1889, p. 175.

[b] Report of Commissioner of Internal Revenue, 1892, p. 205.

[c] Report of the Health Officer, 1893, p. 10.

[d] Report of the Health Officer, 1893, pp. 10, 11. Instructions to Internal Revenue Officers, Series 7, No. 15, Revised, p. 9. August 10, 1893.

> the force or equipment of this division, in connection with the execution of the law and heretofore no such increase has been necessary as no effort has been made to enforce it, consequently few samples have been presented. In consequence of a more active supervision of the milk supply in Washington by the local health authorities, however, a number of samples of milk were presented to this office during August and September, 1893, for decisions as to their adulteration under the provisions of this law. As the time required for the analysis of these samples and for the rendering of expert testimony thereon in court threatened to interfere seriously with the regular work of the division, revised regulations were issued (Series 7, No. 15 revised) providing for the analysis of all such samples by the chemist of the health office of the District of Columbia, under the control and supervision of this office.
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> Reports were accordingly received from this officer from September, 1893, up to the close of the fiscal year, of the analysis of 17 samples—15 of milk, 1 of butter, and 1 of granulated sugar—all of which were decided to be adulterated and so certified to the district attorney for the District of Columbia. I would suggest in this connection that Congress be asked to either provide the facilities necessary for the analysis of all samples in the laboratory of this office, or to so amend the law as to relieve me entirely from any connection with its execution.[a]

After the amendment of the regulations, samples of milk and of other articles of food were collected by agents of the health department and duly analyzed by the chemist. If the result warranted a prosecution, the usual form of application for analysis and certification, provided by the Commissioner of Internal Revenue, was made out in triplicate, the report of the analyst being filled in by the chemist of the health department. After such application had been duly certified, one copy was filed with the district attorney and the prosecution was duly proceeded with. The method was cumbersome and lacked the directness essential to the efficient enforcement of a statute by criminal procedure. Reference of the case to the Commissioner of Internal Revenue was a mere form, since the findings of the chemist of the health department were not verified in the office of the Commissioner by independent analysis, nor was the vendor given an opportunity to be heard. The enactment of this law was, however, of importance as marking a definite effort toward the proper control of the food supply of the District of Columbia and marking also the establishment by regulation of a standard for "whole (pure) milk." The chemist of the board of health had recommended the establishment of such a standard on November 10, 1874;[b] its establishment became an accomplished fact on November 20, 1888.[c] It can hardly be said that any undue haste was displayed with respect to the matter.

The possible relation between the milk supply and the prevalence of typhoid fever in the District of Columbia appears to have been called directly to public attention for the first time, by Prof. J. D.

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[a] Report of Commissioner of Internal Revenue, 1894, page 197.

[b] Report of Board of Health, 1874, p. 207.

[c] Instructions to Internal Revenue Officers, Series 7, No. 15, p. 13.

Hird, chemist of the health department, in his report for the year ending June 30, 1893. Professor Hird said:

> While the effect of lowering the nutritive value of the milk, either by the addition of water or the removal of cream, can be readily comprehended, yet this becomes of secondary importance when we compare this with milk that contains the germs of typhoid and scarlet fever, diphtheria, and tuberculosis in its various forms. Some of these germs grow rapidly in milk without producing any visible effect. The germs of typhoid fever, tuberculosis, and diphtheria may thus grow and be consumed with the milk without our knowledge. The tests ordinarily applied fail to detect the specific germs of these diseases.
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> * * *
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> Some of the common putrefactive bacteria give rise to poisons while growing in this fluid. * * * Cleanliness and care, therefore, become of the most vital importance in the handling of the milk and cans in which the milk is conveyed, while clean stables, pure air and water are as necessary to the animal as to the human being.

The then health officer, Dr. C. M. Hammett, in commenting upon the prevalence of typhoid fever at that time, stated that in some cases the disease had prevailed in families who used water from the same well, and in others where families received their milk from the same cows, and recommended the close and frequent inspection not only of milk and the dairy establishments which supply it, but also of the cattle composing the herd. This subject, said Doctor Hammett, is now receiving the earnest attention of the health department, with a view to the making of an intelligent and effective recommendation to the Commissioners and to Congress.[a]

About July, 1893, Dr. E. C. Schroeder, of the Bureau of Animal Industry, began an investigation into the milk supply of the District of Columbia. Between July 12, of that year, and April 19, 1894, 18 samples of milk were collected, as delivered to private residences or as bought in the stores of this city, and 1 was obtained from a herd in Virginia. Specimens from these 19 samples were injected into the peritoneal cavities of 40 guinea pigs, and in 1 case tuberculosis resulted.[b] The investigation made by Doctor Schroeder included the examination of many dairy cows supplying milk to this city, and out of over 800 examined between 5 and 6 per cent had defective udders.[c] Referring to the investigation then being made, and particularly to the application of the tuberculin test, the Chief of the Bureau of Animal Industry wrote, apparently in the fall of 1894, as follows:

> The testing of cows with tuberculin in the District of Columbia is now in progress, but has been commenced in a small way in order to develop a satisfactory plan of operations. About 125 cows have been tested, and 20 per cent of these were found to be affected.

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[a] Report of the Health Officer, 1893, pp. 9 to 12.

[b] Bureau of Animal Industry, Bulletin No. 7, published in 1894, pp. 77 to 81.

[c] Bureau of Animal Industry, Bulletin No. 7, published in 1894, p. 87.

> I would now recommend a larger force and more vigorous operations. With the present force and facilities, but a few thousand dollars can be used during the year out of the $100,000 appropriated for this purpose. With twice as many men and better facilities for getting over the ground several times as much work can be accomplished.
>
> As tuberculous cows are taken out of the dairies new animals must be put into their places, and it is desirable that these should be tested before they are allowed to enter the stables which have been disinfected and freed from disease. To do this at present the herd inspection must be interrupted, and as the number of inspected herds increases the interruptions will be more frequent, until the herd inspection will be entirely stopped. It is important, therefore, that men should be stationed at the stock yards to test all cows that are brought into the District. This will prevent the further introduction of the disease, and will enable us to keep free from it the herds which have already been inspected.
>
> This work in the District is of great importance, not only as a preventive of disease among consumers of meat and milk, and to guard against the spread of tuberculosis from the District into adjoining States as required by law, but as an experiment to determine various questions relating to the prevalence, recognition, and prevention of tuberculosis as affecting dairy stock. The scientific results of this work are, therefore, of great value to the whole country, while the practical work is of benefit to only a small section. These scientific results are urgently needed in order that the various States may formulate proper measures for protecting their citizens, and for that reason, if for no other, the work should be pressed to early completion.
>
> Under any circumstances only a small part of the amount appropriated can be expended during this fiscal year, as one-third of the year has already elapsed. More rapid work also means more thorough work. There is less opportunity to shift diseased cows from the uninspected to the inspected dairies, and the first inspection should be completed before a second one is necessary.
>
> Detailed recommendations as to requirements will be made from time to time as occasion demands.[a]

No record has been found to show what further recommendations, if any, the Chief of the Bureau of Animal Industry made concerning the application of the tuberculin test to dairy cattle in the District of Columbia, nor to show even why the work that had been begun was abandoned. The reference to "the $100,000 appropriated for this purpose," apparently meaning thereby for the testing of dairy cows in the District of Columbia with tuberculin, is misleading. A careful investigation has failed to show that Congress ever made an appropriation for the purpose named. It is probable that the amount stated was an amount set aside, formally or informally, by the Secretary of Agriculture, for the eradication of tuberculosis from the District, from the gross sum appropriated for the expenses of the Bureau of Animal Industry during the fiscal year 1895. The appropriation act for the year named[b] authorized the

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[a] Tenth and Eleventh Annual Reports, Bureau of Animal Industry, 1893–94. Published in 1896. Pages 32 and 33.

[b] Act of August 8, 1894, 28 Stats., 269.

Secretary of Agriculture to use any part of the money appropriated for the salaries and expenses of the Bureau of Animal Industry, $800,000, that he might deem necessary or expedient and in such manner as he might think best, to prevent the spread of pleuropneumonia, tuberculosis, sheep scab, and other diseases of animals, and to expend any part of the appropriation in the purchase and destruction of diseased or exposed animals and the quarantine of the same whenever in his judgment it might be necessary to prevent the spread of such diseases from one State to another. The authority of the Secretary of Agriculture to set aside $100,000 for the eradication of tuberculosis from the District of Columbia under the terms of this act seems to have been ample. The fact that this appropriation was made to apply specifically to tuberculosis, whereas preceding appropriation acts had not named this disease, suggests that Congress may have had in mind when it was made the inauguration of active operations toward the eradication of tuberculosis.

Whether the work done by the Bureau of Animal Industry in the District of Columbia during 1893 and 1894 was prompted by the then recent discovery of tuberculin and the announcement of its properties and uses, or was begun because of the general sanitary awakening that had been brought about by the dread lest Asiatic cholera, then prevailing in certain parts of Europe, should gain entrance and foothold in this country, the record does not disclose. The latter circumstance, however, was a potent factor in creating a popular sentiment favorable to sanitary improvement. In this District an organization denominated the Sanitary League was formed and popular lectures on subjects related to hygiene and sanitation were held. In the fall of the year appeared the first report of the then newly appointed chemist of the health department showing the utter inadequacy of existing legislation for the protection of the milk supply.[a] The time for action had come. The Commissioners asked the cooperation of the Medical Society of the District of Columbia in framing necessary legislation, and the society promptly appointed a committee, consisting of Drs. C. H. A. Kleinschmidt, S. S. Adams, and W. C. Woodward, to investigate the subject and to report to the society. The report of this committee was submitted on June 13, 1894, and embodied a draft of the proposed bill. The report was approved, and the proposed bill forwarded to the Commissioners, but the session of Congress had advanced so far that favorable action was impossible. The legislation suggested was later approved by Dr. D. E. Salmon then Chief of the Bureau of Animal Industry, Department of Agriculture. It became a law on March 2, 1895, under the title of "An act to regulate the sale of milk in the District of Columbia, and for other purposes,"[a] only two amendments tending seriously to impair its usefulness from an administrative standpoint having been made. The food inspectors in the service of the board of health had, in October, 1873, recommended that persons selling milk in the District of Columbia be required to obtain permits in order to obtain a basis for the proper inspection of places where milk was produced and sold, and of the cattle from which it was drawn. The milk law of March 2, 1895, had now accomplished that end.

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[a] Report of the Health Officer, 1893, pages 9 to 12.

The new law marked a departure in milk legislation from established lines. Theretofore it had been deemed sufficient to examine milk as it appeared in the market. Now it was proposed to begin at the cow. Previously it had been regarded as beyond the power of the community to go outside of its territorial limits to control the methods employed in the production of its food supply. Now it was proposed to say to the producer, no matter where located, " The milk sold in our jurisdiction must come from places that conform to certain requirements, as determined by inspection by our own agents. If you wish to sell milk of this kind, and none other can be sold in our city, we will, if you desire and request it, inspect your establishment for you." It must be admitted that the District of Columbia was in one way peculiarly well situated to attempt such a departure from established law and practice; for while by far the larger part of its milk supply comes from the States of Maryland and Virginia, yet any law that might be enacted must emanate from Congress, vested not only with the right to exercise exclusive legislation in all cases whatsoever over the District of Columbia, but also with the right to regulate interstate commerce. Such a statute might be enacted by that body therefore, with less likelihood of attack than if enacted by a State legislature or by a municipal council.

The milk law enacted in 1895 made it the duty of the health officer of the District of Columbia, under direction of the Commissioners, to make and enforce regulations to secure proper water supply, drainage, ventilation, air space, floor space, and cleaning of all dairies and dairy farms within said District; to secure the isolation of cattle suffering from any contagious disease, and to carry into effect the provisions of the act. These regulations were duly made, and under date of June 26, 1895, were approved by the Commissioners.[b] It was the belief of the health officer that these regulations should be specific rather than general, and as originally drafted they were of the former character. The then attorney for the District, however, entertained a different opinion, and in deference to his more extended experience with respect to such matters the regulations were reduced to their now somewhat general form, whether wisely or unwisely may properly be questioned. They have been amended from time to time and in the form in which they now exist appear elsewhere in this report. So also do various extracts from the building and police regulations bearing directly upon the construction and management of dairies.[a]

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[a] See page 808 for full text of law.

[b] Report of the Health Officer, 1896, page 256.

The milk law of 1895 represented at the time of its enactment a departure from established precedent. It was a more or less experimental measure, and therefore it could not be expected that it would be found to meet perfectly all the requirements of the service when put into operation. Experience soon revealed defects, and efforts were promptly begun to correct them. As early as December 15, 1896, bills were introduced into Congress for that purpose, and legislation to accomplish the desired end has been pending before that body almost continuously ever since. The result, however, has not been encouraging. Bills introduced on the recommendation of the health officer, and receiving the indorsement of the Commissioners and of the Medical Society of the District of Columbia, have been uniformly opposed by milk interests. Unfortunately, it has been impossible in the drafting of legislation to deal with the milk interests as with a unit. The men who are engaged in the production of milk and in some cases in its distribution, and those who are engaged solely in buying milk and delivering it to the consumer, are too numerous and too widely scattered, and their interests are too diverse, to have enabled them to come together in a compact organization which might be reached as a whole, through its meetings or through any trade or society journal. It has been impossible for the health officer to submit to the Commissioners or for the Commissioners to submit to Congress any bill to regulate the sale of milk in the District with the assurance that it would not meet with more or less formidable opposition from persons interested in the production and sale of milk, either individually or as an organization. The fight to obtain better legislation to regulate the sale of milk has always been carried to the committee room, at the Capitol, and the fight has always been lost.

While it has been impossible to obtain needed amendments to the act of March 2, 1895, regulating the sale of milk, other legislation has been enacted that has modified the practice of the health department with respect to the supervision of the milk supply and the maintenance of the milk-inspection service. By the act of February 17, 1898, entitled "An act relating to the adulteration of foods and drugs in the District of Columbia,"[b] the required chemical composition of milk was altered so as to raise the minimum allowable amount of butter fat and of total solids in whole milk from 3 per cent and 12 per cent, respectively, as fixed by the milk law of March 2, 1895, to 3½ per cent and 12½ per cent, respectively. By the same act a standard for the chemical composition of cream was fixed, requiring a minimum of 20 per cent butter fat, and this act changed generally the method of procuring samples. By the act making appropriations for the expenses of the District of Columbia, approved April 27, 1904, the following provisos bearing upon the enforcement of the laws and regulations relating to the sale of milk were enacted, and they were repeated in each of the District appropriation bills passed until that of May 26, 1908:

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[a] See page 819.

[b] See page 810 for full text of this act.

> *Provided*, That no officer or employee of the health department shall, during his continuance in office, serve in his private capacity for fee, gift, or reward any person licensed to keep or maintain a dairy or dairy farm in said District, or to bring or to send milk into the said District, or any person who has applied or is about to apply for such license, or any manufacturer or dealer in foods, drugs, or disinfectants, or similar materials: *Provided, further*, That every place where milk is sold shall be deemed a dairy under the law for purposes of inspection.[a]

The first of these provisos was inserted in the then pending appropriation bill in connection with a proposed increase in the salaries of the employees in the service of the health department whom the proviso was most likely to affect—that is, the inspectors of dairy farms. The increase in salaries was not made, but the proviso was allowed to remain. The effect was to deprive the inspectors of dairy farms of certain of the opportunities that they had previously had to add to the scant incomes that their official positions provided. Of these opportunities they had theretofore been allowed to avail themselves from time to time as demands were made for their professional services, and they had done so without criticism or complaint. The principle laid down in this proviso is, however, recognized as eminently wise and proper, and yet it would seem that the fact should be recognized that its enactment into law reduced the possible incomes from private sources of the employees who come within its scope, and that due compensation should be made because of that fact. The second proviso has unfortunately failed entirely to accomplish the purpose for which presumably it was enacted—that is, to require every vendor of milk to provide himself with facilities for storing and distributing it similar to those required of proprietors of licensed dairies under like circumstances. The failure of the proviso has been due to the insertion of the words, "for purposes of inspection," the effect of which has been to limit the purpose for which places can be regarded as dairies, in which places milk is sold merely as an incident to some other business, to but one single thing, inspection. Neither for purpose of licensing or construction or management do the dairy regulations apply.

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[a] 33 Stats., 383.

The most recent legislation by Congress relating to the sale of milk in the District was enacted on February 27, 1907, under the title "An act to amend section eight hundred and seventy-eight of the Code of Law for the District of Columbia."[a] The purpose of the amendment was to extend to dairymen the right, enjoyed by dealers in other beverages, to register with the clerk of the supreme court of the District of Columbia distinctive marks for the identification of the vessels in which deliveries are made, and thus to secure exclusive right to the use of such vessels, under pain of fine or imprisonment imposed on any person trespassing against such right.[b] The general statute commonly known as the pure food and drugs act,[c] June 30, 1906, by its terms covers much of the field covered by the District milk act of March 2, 1895, and the District pure-food act of February 17, 1898; so much so, in fact, that in a prosecution under the act last named, based upon the sale of adulterated milk, its validity was attacked on the ground that it had been repealed by implication through the enactment of the federal pure food and drugs act. The police court decided that the local statute had been so repealed and acquitted the defendant, but upon appeal by the District[d] the case was disposed of without the court finding it necessary to decide whether the earlier act had or had not been repealed. The court of appeals, however, referring to the question of repeal, said:

> The question as to the entire repeal of the earlier act, the operation of which was confined to the District of Columbia by the later general law, is one of great importance that ought to be authoritatively settled. It is unfortunate, therefore, that the police court did not content itself with quashing the information and dismissing the prosecution, in accordance with its view of the law, without going further and adjudging the defendant not guilty. While it seems probable that the court took an erroneous view of the law, we are without jurisdiction to express an opinion upon the question, by reason of the judgment actually rendered.

Since the rendering of the decision from which the foregoing quotation is taken, the health department has prosecuted vendors of adulterated milk and cream under either statute, according as prosecution under the one or the other was most convenient or seemed likely to yield the better results, and the question as to whether there has or has not been a repeal of the District pure-food law of February 17, 1898, has not been settled.

The results of the extensive investigations recently made by the Federal Department of Agriculture into the condition of the dairy farms and dairies supplying milk to the District, and of the milk sold here, have not yet been officially published. The results of the work done by the Public Health and Marine-Hospital Service during the summer of 1906, in connection with its investigation into the cause of the undue prevalence of typhoid fever, appear at length in one of the recent bulletins published by that service.[a] All that is of general interest of the report of the conference called by the Commissioners of the District on March 30, 1907, to determine what should be done to improve the milk supply has been printed in certain circulars lately issued by the Department of Agriculture.[b] As valuable as this work has been, yet, in view of what has already been published concerning it and in view of the probable issue of further bulletins relating thereto, all of which are or will be available to those interested in the subject, it does not seem expedient to give any detailed account of it here.

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[a] 34 Stats., 1006.

[b] See page 818 for full text of this law.

[c] 34 Stats., 768.

[d] D. C. *v.* Burns, 32 Appeals, D. C., 203.

It could not be expected that with any reasonably rigid enforcement of the laws and regulations relating to the production and sale of milk, and the manufacture and sale of foods generally, over any considerable period, questions of construction would not arise and be submitted to the courts for adjudication. The first case that seems to be at all material to the purposes of this report is District of Columbia *v.* Lynham,[c] decided February 7, 1900. The case arose under the provisions of an act relating to the adulteration of foods and drugs in the District of Columbia, approved February 17, 1898,[d] and the particular question submitted for decision was whether the defendant, a druggist, charged with the sale of a drug adulterated within the meaning of the act, was entitled to acquittal upon showing that he was at the time of sale ignorant of the composition of the substance sold. The court says:

> In the trial of a prosecution under this statute it is incumbent upon the District of Columbia, in whose name the prosecution is conducted, to prove the sale and delivery of the medicine or drug by the defendant, or his possession thereof for purpose of sale, and that the same was adulterated within the meaning of the statute. The prosecution upon such proof makes out a prima facie case of guilt against the defendant; and it is no defense for the defendant to show simply that he was at the time of sale, or of possession for sale, ignorant of the fact of such adulteration of the drug or medicine. He must know what he sells, or proposes to sell, and that it conforms to the standard prescribed by law. As a registered druggist, he holds himself out to the public as being sufficiently skilled to know and understand of what constituents or ingredients the drugs and medicines that he offers for sale are composed, and especially in respect to all such drugs and medicines as are recognized and described in the Pharma-

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[a] Public Health and Marine-Hospital Service, Hygienic Laboratory. Bulletin No. 35. February, 1907.

[b] Bureau of Animal Industry, Circular 111, issued June 22, 1907. Circular 114, issued August 20, 1907.

[c] 16 Appeals, D. C., 185.

[d] 30 Stats., 246.

copœia. It is not in his mouth to say, when it is shown that the drug was impure or adulterated at the time of sale, that he was ignorant of the fact. If such defense could be allowed, there would be no protection to the public against impurities and adulterations of drugs and foods.

The second case to be decided, Weigand *v.* District of Columbia,[a] decided November 5, 1903, involved, among other things, the application of the principle enunciated in District of Columbia *v.* Lynham (supra). It involved also the construction of "An act to regulate the sale of milk in the District of Columbia, and for other purposes," approved March 2, 1895,[b] and of "An act relating to the adulteration of foods and drugs in the District of Columbia," approved February 17, 1898.[c] Weigand had been convicted in the police court of selling adulterated milk, to wit, milk containing less than 3½ per cent of butter fat. He had sought to show by evidence, and to have the jury instructed, that the provision of the act of Congress of 1898, prescribing the standard of milk for sale in the District of Columbia, was unreasonable and oppressive, and therefore void. But the court said:

> To declare an act of Congress unreasonable and oppressive, and therefore void, is a power that the courts can not exercise, except where the provision of the statute is shown to be plainly violative of some provision of the Constitution. The subject-matter of the act of 1898 is plainly within the power of Congress, and the courts can not amend or modify any of the provisions of that act so as to bring them within what may seem to be reasonable bounds. They can not examine questions as expedient or inexpedient, as politic or impolitic. Considerations of that nature must, in general, be addressed to the legislature. Questions of policy determined there are conclusive with the courts. (License cases, 5 Wall., 462, 475.) If, by the plain words of an act of Congress, an impossible thing was required to be done, or some thing done in an impossible manner (if such legislation could be rationally supposed to occur), in such case the courts would have no alternative but to declare the statute to be incapable of enforcement in the particular case. But statutes are not to be declared void because of difficulty of construction, or because of apparent hardship in their application; nor are the plain words of a statute to be refused their application upon any theory that a more reasonable provision could have been adopted for the state of case presented. All statutes must receive a sensible construction, such as will effectuate the legislative intention, and, if possible, so as to avoid an unjust or an absurd conclusion. (Law Ow Bow *v.* United States, 144 U. S., 47, 59; Hawaii *v.* Mankichi, 190 U. S., 213.) It is true a municipal ordinance professed to be passed under a general or implied power given by a statute must be reasonable and lawful, and not oppressive, and if it be not so it will be declared void. But this is upon the presumption that the legislature did not intend by the general terms of the statute to authorize the making of such an ordinance. (1 Dill., Mun. Corp., sec. 319; Cooley, Const. Lim., 192, 193, 6th ed.) And it has therefore been held that an ordinance can not be held to be unreasonable and void which is expressly authorized by the legislature. (Coal Float *v.* The City of Jefferson, 112 Ind., 15; Cooley, Const. Lim., 241.)

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[a] 22 Appeals, D. C., 559.

[b] 28 Stats., 709.

[c] 30 Stats., 246.

> In this case the offer was made to show, and the court was requested to declare, not that the act of Congress required milk to conform to an impossible standard or test, or that the milk offered for sale should contain constituents that nature did not supply, but that the standard prescribed was unreasonably high, and could not by ordinary care be maintained through all seasons of the year. There may be difficulty in keeping up the standard throughout the year, and more expense and greater effort may be required at some seasons of the year than at others. But the very object of the statute was to require this more than ordinary expense and labor, on the part of the owner of cows, to keep up and maintain the prescribed standard of milk when necessary; and this is accomplished by proper care of and food supplied to the animals producing the milk. For it is well known that the quality and richness of milk depend largely upon the condition of the animal, the care with which it is kept, and the kind and quantity of food supplied to it. It is not attempted to be shown that 3½ per cent of fat, as a constituent of good milk, is greater than can be supplied by proper care of, and good and abundant food supplied to, the cows. If the proposition of the defendant were sustained, the question of the reasonableness of the statute would be one of fact for the jury, and we should likely have different juries determining the question in different ways. We think the court was clearly right in its ruling upon this question, and in holding that the question, whether the standard of milk prescribed by the statute was reasonable or not, was not open to inquiry on the trial.

In the police court Weigand had been denied, too, the right to show by evidence the specific purpose for which he had in his possession the milk from which the sample was taken, but the appellate court found no error in the exclusion of such evidence, saying:

> But under section 3 of the act of 1898 the question is whether the sale was made of the article, which was in fact under the standard prescribed by the law. The party making the sale is bound at his peril to know what he is selling, and, to keep within the law, he must know that the article complies with the standard of excellence and purity prescribed by the law. Unless this be so, it would be very difficult, if not impossible, ever to convict a party of a violation of the law. And for the same reason the court below was right in refusing to allow the defendant to introduce evidence to show for what purpose he had kept the milk on hand, that being entirely immaterial, if he sold the milk that did not bear the test prescribed.

The other questions decided by Weigand *v.* District of Columbia are not material for present purposes.

The third case, District of Columbia *v.* Garrison,[a] decided May 23, 1905, arose under the provisions of "An act relating to the adulteration of foods and drugs in the District of Columbia," approved February 17, 1898,[b] and required the determination of the extent of the right of the inspectors in the service of the health department to purchase samples of food for analysis, under section 6 of the act, which is as follows:

> That every person offering for sale or delivering to any purchaser any drug or article of food included in the provisions of this act shall furnish to any analyst or other officer or agent of the health department, who shall apply to

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[a] 25 Appeals, D. C., 563.

[b] 30 Stats., 246.

> him for the purpose and shall tender him the value of the same, a sample sufficient for the purpose of analysis of any such drug or article of food which is in his possession.

The defendant had declined to sell to an inspector one-half pint of milk upon the tender of the usual price therefor, 2 cents, but had offered to sell one entire pint for 4 cents, alleging that he sold milk only in the original packages in which he received it, that he had no package containing less than 1 pint, and that if he sold a half-pint from such a package the remaining half pint would represent a loss to him, since his customers knew that he did not sell milk in quantities less than a pint, and that therefore he had no demand for half pints. The police court having sustained the position taken by the defendant, an appeal was taken on the recommendation of the health officer. It was apparent that if one dealer were permitted to refuse, on the grounds taken by the defendant in this case, to sell less than a pint, another might fairly claim the right to refuse, on the same grounds, to sell less than a quart, and so on; that if inspectors were required to accept and to bring to the laboratory samples as large as 1 quart, or even as large as 1 pint, their return trips to the laboratory from the field must be correspondingly more frequent and their working capacity correspondingly diminished, and that if, on the other hand, the inspector undertook to mix the pint of milk thoroughly in the pint jar in which it was delivered to him or to mix a quart of milk in a quart jar, and to abstract therefrom a sample for analysis, the fairness of the sample might readily be called into question in event of prosecution. Moreover, the act under which the prosecution was brought limited the size of the sample which the inspector might demand to "a sample sufficient for the purpose of analysis;" and one-half pint of milk was sufficient, the inspector could not demand more, and it was not apparent why he should be required to accept more; and the court of appeals had already declared (Weigand *v.* D. C., 22 Appeals, D. C., 559) that the subject-matter of the act of 1898, under which act the right to purchase the sample had been claimed, was plainly within the power of Congress, and that the courts could not amend or modify any of the provisions of that act so as to bring them within what might seem to be reasonable bounds; that they could not examine questions as expedient or inexpedient, as politic or impolitic.

The court of appeals, however, after denying that any principle was involved in the case, and after a scathing criticism of the health department for the course it had pursued, said:

> A reasonable sample is what is required by the act of Congress. Under the circumstances of this case a pint was a reasonable sample, and a half pint was not such a reasonable sample. The appellee was fully within his right, and fully performed his duty in tendering the former; the inspector was

wholly at fault in demanding the latter and insisting upon it against the will of the appellee.

What may or may not be a reasonable sample is a question for which perhaps no positive rule can be laid down applicable to all cases. This is not for the determination exclusively either of the inspector or the dealer. The act requires that it shall be "sufficient for the purpose of analysis," but it is not competent for the inspector to require, because he thinks a half pint of milk sufficient to enable him to make a satisfactory analysis of such milk, that therefore the dealer must sell him such half pint, when thereby the value of another half pint would be destroyed or lost to the dealer, and the dealer is willing to sell an entire pint at an additional cost of merely 2 cents to the inspector.

II. ORGANIZATION AND DUTIES OF THE MILK-INSPECTION SERVICE.

Nothing worthy of the name of a milk-inspection service can be said to have existed in the District of Columbia prior to the passage of the act of Congress of March 2, 1895, for the regulation of the sale of milk. Not even, in fact, was a milk-inspection service established by that act. The necessary authority was conferred, but no special inspectors or funds were provided through which to exercise that authority, and the health officer in the execution and enforcement of the law had to rely upon the inspectors already provided for the sanitary and food inspection service generally, and upon the allotment made by the Commissioners for the contingent expenses of the health department from the general appropriation for that purpose. The health department had no veterinary surgeon in its employ, and for such assistance as was needed in the way of inspections requiring the education and training of a veterinarian had to rely upon the veterinary surgeon employed by the District government, at a salary of $400 per annum, for all departments of the District government. The situation of the health department under the circumstances was most unfortunate; errors that were made in the early days of the service because of an insufficient and untrained inspection force have come up from time to time to embarrass the department, and have been corrected with difficulty, if at all.

The conditions found as the result of such early inspections as were made showed in many cases entire ignorance of even the most rudimentary sanitary principles connected with the production and marketing of milk. The stables were small, poorly lighted, and poorly drained. Many of the producers of milk had no idea of the importance of cooling it immediately after milking, and sometimes did not hesitate to store it in living rooms and kitchens.[a] The condition of the cattle can be best understood from the statement made by Doctor Schroeder, of the Bureau of Animal Industry, as the result of the examination of over 800 of our dairy cattle during 1893 and 1894, that between 5 and 6 per cent had defective udders.[a] Those who are sometimes inclined now to criticise existing conditions would be able to do so more intelligently and fairly were they familiar with the conditions found in the early days of the milk-inspection service. No good purpose would be served by here recounting at length the various steps taken for the improvement of the service that was established. It is sufficient to say that improvement generally has been possible only as appropriations have been made by Congress to bring them about, and that requests for such appropriations have not always met with favorable response. A statement showing the growth of the milk-inspection service is appended.[b] It is enough here to describe the milk-inspection service as it now exists.

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[a] Report of the Health Officer, 1896, page 21.

The law regulating the sale of milk in the District of Columbia prohibits the maintenance of a dairy or a dairy farm within the District without a permit from the health officer. It does not define what a dairy or a dairy farm is. In practice, the health department has held the term "dairy" to mean the business arising from milk products, or a store devoted to the sale of milk and its products, and has held the term "dairy farm" to mean any premises upon which milk is produced for sale. Any place where milk is sold is regarded by the law of the District as a dairy for purposes of inspection, but not for other purposes.[c] Permits issued under the provisions of the act of March 2, 1895, have been issued, therefore, in three distinct series: First, permits to maintain dairies within the District of Columbia; second, permits to maintain dairy farms within the District of Columbia; third, permits to bring or to send milk into the District of Columbia. In order that the records of the health department might show the number and the location of places from which milk is distributed as distinguished from the number and location of places where milk is produced, persons maintaining dairy farms within the District of Columbia and distributing milk directly to consumers have been regarded as maintaining dairies as well as dairy farms and have been required to obtain a permit for each purpose. A similar practice has been established with respect to persons maintaining dairy farms in adjacent States and distributing the milk directly to consumers within the District; they have been required to take out permits not merely to bring or send milk into the District, but also to obtain permits to maintain dairies within the District. In the issue of permits to maintain dairies to persons already authorized to bring or to send milk into the District they have not been required to maintain fixed places of business within the District, but their dairy farms have been regarded as the points of distribution. Copies of the forms now in use for application and for permits are appended.[a]

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[a] Bureau of Animal Industry, Bulletin No. 7, published in 1894, page 87.

[b] See page 792.

[c] See page 762.

The inspection service is naturally divided into two branches: On the one hand the inspection of dairy farms and on the other the inspection of dairies. But whenever a permit is issued for the maintenance of a dairy on a licensed dairy farm, either within or without the District, then inspections of the dairy are intrusted solely to the inspector of dairy farms already having the premises under his supervision, and the inspector of dairies is not required to visit the premises.

The inspection of dairy farms requires not only a knowledge of the conditions under which milk should be produced, but also a knowledge of cattle, their selection, their feeding, their general management, and their diseases. Such work is therefore best intrusted to veterinary surgeons, and inspectors of this class have always been required to have had a proper veterinary training before appointment. The inspection of dairies, places from which milk is sold at retail, requires, however, only a good working knowledge of the sanitary principles underlying the handling of milk. For these purposes, therefore, and for the collection of samples of milk, men have been selected because of their general qualifications, and have not been required to have special training or experience with respect to the milk business. In certain cases, in making appointments of men to be assigned to the inspection of dairies, weight was allowed to the fact that the applicants had been engaged in the dairy business, but the result can not be said to have justified the anticipations of the department.

The knowledge and experience of the inspectors of dairy farms and the inspectors of dairies must be supplemented by knowledge of the chemical composition of milk and of milk products, and of water, and by a practical acquaintance with the methods of analyzing these substances. A chemist is employed for that purpose. The knowledge of these inspectors ought to be supplemented by a knowledge of the bacteriology of milk and of milk products, and of water, and by ability to analyze them microscopically and bacteriologically, but appeals from the health officer for an appropriation for the establishment and maintenance of a bacteriological laboratory, with a competent bacteriologist in charge of it, have not yet been favorably acted upon. For the proper supervision and control of the work of the inspectors of dairy farms, and of dairies, and of the work of the chemist, records must be kept and a very considerable volume of correspondence handled, imposing upon the department a large amount of clerical work. The amount of correspondence arising in connection with the milk-inspection service is very large, because so many of the persons producing milk for sale within the District and whose premises are under inspection reside at points more or less remote from the city. The milk-inspection service is organized, in order to meet the conditions described above, as follows: (*a*) Supervision; (*b*) inspection of dairy farms; (*c*) inspection of dairies; (*d*) inspection of milk.

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[a] See page 793.

SUPERVISION.

The magnitude and importance of the milk-inspection service amply justifies the employment of a chief inspector, to devote his time exclusively to supervising the work. The work to be done, however, calls for special qualifications, and men possessing such qualifications can not be induced to enter the service of the Government and abandon all else, especially with no assured tenure of office, unless they are reasonably well paid. And no provision has ever been made for so compensating any such officer. As a matter of expediency, therefore, the supervision of this service is divided. The chief inspector of the health department is responsible for the sanitary condition of places where milk is sold within the District and for the collection and analysis of samples of milk and cream. The supervision of the places where milk is produced, the dairy farms, whether within or without the District, and the keeping of the records pertaining to such places, is intrusted to a sanitary and food inspector detailed for that purpose.

The inspector in charge of the contagious-disease service is required, in investigating such cases of typhoid fever, scarlet fever, and diphtheria as come to his notice, to ascertain whether they have possibly had their origin in the milk supply, and on the slightest suspicion to cooperate with the chief inspector and the inspector in charge of the dairy-farm service in ascertaining the exact facts of the case, and, under direction of the health officer, in taking such remedial action, if any, as may be found advisable.

The inspector in charge of the dairy-farm service is supposed to devote at least one day each week to the personal inspection of the dairy farms under his supervision, and the chief inspector or his assistant is expected to be in the field at least one day each week looking after the work of the general inspection service, which is under his immediate control and which includes the inspection of dairies. All papers relating to any given dairy or dairy farm, including the original report made by the dairy farm inspectors and carbon copies of score cards and of notices served, and copies of letters received and letters sent, are filed in jackets in such manner with the papers relating to any one given dairy or dairy farm, and are kept together so as to facilitate ready reference. These records are open to the public, and persons desirous of knowing the standing of any dairy or dairy farm are given every facility to examine them and are urged to do so.

INSPECTION OF DAIRY FARMS.

The total number of farms now licensed to produce milk in this jurisdiction or to send milk into it from the adjacent States, Maryland and Virginia, is 864. Milk is being shipped into the District from 18 farms in Pennsylvania, 18 in New York, and 12 in New Jersey, without licenses, by virtue of the provision in the law which authorizes the shipment of milk immediately after the filing of an application and until that application has been acted upon by the health officer. The funds available for the inspection of dairy farms have not been sufficient to permit the systematic inspection of farms in New York and Pennsylvania, and therefore action by the health officer has been indefinitely suspended. The applications that have been filed present, of course, prima facie cases of full compliance with the laws and regulations of the District, and licenses might be lawfully issued. Experience has demonstrated, however, that the evidence presented in an application is not always to be relied upon, and therefore, pending the making of provisions for the regular inspection of these places, the course just set forth has been adopted.

The territory under actual inspection is divided into six districts and one inspector assigned to each. Prior to September 20, 1907, there were but five districts, only five inspectors being then available for the inspection of dairy farms. The result was that in one district a considerable number of farms could not be inspected during the entire year.

In order that an inspector of dairy farms may discharge his duties, it is necessary that he be provided with means of transportation. For this purpose each inspector of dairy farms is allowed by the District government $365 per annum to enable him to maintain a horse and vehicle for his official use. When an inspector is required to visit dairy farms beyond a driving distance from his place of residence, he is allowed actual traveling expenses, payment being made monthly on itemized vouchers, duly sworn to by the inspector and approved by the health officer.

Notwithstanding the use of a horse and vehicle and the making of allowance for traveling expenses, the amount of work done by an inspector of dairy farms is seriously curtailed by the distance which he has to travel between the places which he inspects. This is true particularly in the more remote portions of the country from which the milk supply is derived, since dairy farms in such regions are few and far between. A certain part of the inspector's time, too, not infrequently goes for naught during the summer season, when, if the cattle are to be examined, he must wait for them to be brought up from the pasture. And at any time of the year a careful inspection of a dairy herd of any considerable size, including, as it must, an examination of the udders and lungs, of itself consumes a considerable time. In the most thickly settled parts of an inspection district an inspector may make six or eight inspections in a day. In remote regions he may be able to make but two and possibly only one. The records of the department show that the average number of dairy farms visited daily by one inspector during the fiscal year ended June 30, 1909, was 2.7, the computation being based upon the work of the six inspectors then on duty and on the actual number of days worked by the entire force on the inspection of dairy farms, viz, 1,589. The average frequency with which each of the 864 dairy farms actually inspected was visited during the period named was 4.9.

An inspector assigned to an outlying district is required to live within the territory under his supervision. Thus, one inspector resides at Leesburg, Va., another at Germantown, Md., and a third at Frederick, Md. The inspectors having supervision of the three districts adjacent to the city, and in fact extending within it, reside in Washington. The number of dairy farms now under the inspection of each of these inspectors is as follows:

Washington district No. 1	141
Washington district No. 2	117
Washington district No. 3	125
Leesburg district	136
Germantown district	182
Frederick district	163

The local inspectors of dairy farms ordinarily visit the health office each morning to file reports of the preceding day's work and to receive instructions as to their duties for the day. If, however, an inspector expects to go a long distance during the day, in order to visit an outlying farm, this customary visit may be omitted. The inspectors residing at Leesburg, Germantown, and Frederick, under ordinary circumstances, visit the health office but once each month, forwarding to the health officer by mail daily such reports as may be necessary, and receiving their instructions in like manner. In any event, each day, either after visiting the health office, if a visit be required, or without such a visit if none be necessary, the inspector proceeds to examine certain of the dairy farms lying within the territory assigned to him. In determining which of these farms to visit he is guided by instructions from the health officer, by outstanding notices which he has served, and by a general knowledge of the condition of the farms within his district. It is just here that the services of a bacteriologist are most needed in connection with milk-inspection work. The bacteriological examination of milk as it reaches the city would show the location of the farms sending in persistently milk containing relatively large numbers of bacteria as compared with the general milk supply. The occurrence of such high bacterial counts being a sure indication of faulty methods of milking or of handling milk, inspection could be directed against the offending farms, farms having good records being inspected with less frequency, if necessary, to permit this to be done effectively.

Having arrived at the farm, the inspector investigates the condition of the premises and of the dairy apparatus and utensils used in connection therewith, and examines into the condition and health of the cattle. He must rely upon his own powers of observation so far as is possible. When this is not possible he must learn what can be learned by inquiry. For instance: A knowledge of the prevailing method of milking and of the promptness and thoroughness of cooling milk is very essential in every case, but in view of the distances between dairy farms the inspector commonly can not visit on a single day, at milking time, more than one, and rarely more than two farms; he, therefore, can not always obtain information with respect to this matter by his own observation, but must depend upon the statements of the farmer and of the farmer's help with respect to it. The same is true to an even greater extent with respect to the possible presence on the dairy farm of communicable diseases, such as typhoid fever or scarlet fever. Never is the veterinary inspector able to report positively, solely as the result of his own observation, the existence of any such disease on the premises; he must rely on such information as he is able to obtain from the farmer and those about the place, and to pick up from others in the neighborhood.

Should an inspector of dairy farms, as the result of his examination of the premises and of the cattle, find conditions in violation of the regulations governing such matters, his course is governed to a considerable extent by the location of the farm, whether it be within or without the District of Columbia. If the farm be within the District the only means available for the enforcement of compliance with law is prosecution in the police court; the offense is committed within the District, and the proprietor of the farm who is responsible for such offense is within the reach of ordinary criminal process. On the other hand, if the farm is located outside of the District, the offense, in so far as relates to the condition of the premises and cattle, is committed in another jurisdiction and not only is the offender beyond the reach of any ordinary criminal process issuing out of the courts of the District, but the condition of the farm, located as it is in another State, is not and can not be in violation of the laws of the District of Columbia so as to render the proprietor criminally liable. His offense begins when he brings or sends milk into the District without a permit to do so; but his permit, if he has one, is by law conditioned upon the management of his farm in accordance with the laws of the District in so far as those laws do not conflict with state laws; and, therefore, if his farm presents circumstances in violation of law, the conditions upon which his permit was issued have been violated by him, and his permit becomes voidable at the election of the health officer, even if it does not become actually void. When a dairy farm is located beyond the limits of the District, compliance with District laws and regulations is enforced, therefore, not by immediate criminal prosecution, but by the cancellation of the permit, if necessary, and then if the milk from the tabooed farm is brought into the District, by criminal prosecution of those responsible for the importation of the milk—the farmer who sends it in, if he can be found in the jurisdiction, or the dealer who contracts with the farmer to have the law violated and cooperates with him in its violation, or both farmer and dealer. Unless, however, the violation of the regulations is extremely grave, or unless similar offenses have been of repeated occurrence, summary action is not taken, but the offender is given an opportunity to correct conditions, or at least given an opportunity, if he be a nonresident, of showing cause why his permit should not be canceled. If he be a resident of the District, even after final action by the health officer, he has his day in court. What has been said with reference to the cancellation of permits has had reference to cancellation because of the existence of insanitary conditions. Permits may be suspended or revoked, whether the farm be within or without the District, if the milk supply therefrom is exposed to infection by certain contagious diseases, but this is a matter that will be considered later.[a]

If, then, an inspector of dairy farms has found conditions that must be corrected, he serves a notice on the responsible person requiring him to correct them within a specified period, or if the conditions are particularly bad, he may, if the farm is within the District, recommend immediate prosecution in the police court, or, if the farm is located outside of the District, he may serve at once a notice requiring the licensee to show cause on or before a given day, to the satisfaction of the health officer, why his permit should not be canceled.[b] If the farm be one for which no license has been issued, but for which application is pending, then, if the circumstances warrant such action, the inspector may recommend the immediate rejection of the application. Any notice prepared by the inspector is written in duplicate by means of carbon paper, the original being left on the premises and the carbon copy being forwarded to the health officer so as to reach him on the day following the day of service.

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[a] See page 783.

[b] For forms of notice, see page 801.

Inspectors have uniformly endeavored to cooperate with the farmer as much as possible toward securing the improvement of his premises. As an incident to the inspection of dairy farms, and in order that the dairy farmer and the health officer might be advised as fully and intelligently as possible of the inspectors' findings, the health officer devised some time ago[a] a system of scoring dairy farms, a feature of dairy-farm inspection which has since been widely adopted. It was deemed desirable, whether a notice was served or not, to inform the farmer as accurately as possible, at each visit, of the judgment of the inspector with reference to his establishment. A certain number of points was, therefore, allowed for each general feature of the dairy farm and the inspector required to grade the establishment accordingly. The form for scoring shows the maximum number of points attainable and the number allowed by the inspector, and is printed so that by means of carbon paper a duplicate copy can be produced without additional labor. The original is left with the farmer for his information and guidance, and the carbon copy is forwarded to the health officer with the daily report of the inspector of dairy farms. With respect to the scoring of dairy farms, however, this may be fairly said: That no one can interpret the meaning of another's score card unless he knows the principle upon which the rating has been made. It is possible either to fix an absolutely ideal standard of perfection and to score every feature of the establishment on that basis or to fix a reasonable standard, having in view the state of the dairy industry, either generally or in the vicinity and at the time when the scoring is to be done. The former method will give low scores; the latter will give higher ones. It can not be said that either method is wrong, and possibly, having in view the future state of the dairy art that is so devoutly to be hoped for, the former method is preferable. It is essential now, however, only that when we undertake to determine the significance of the scores of dairy farms we know which method has been followed. In one place, apparently under the former method of scoring, 30 points out of a possible 100 has been regarded as a fair passing mark.[b] Under the other method 70 would be none too high.

Recently the Department of Agriculture has applied the tuberculin test to a considerable number of the dairy herds supplying milk to the District. This test has been applied under an "agreement" between the Department and the farmer, the latter undertaking to comply with certain conditions in consideration of the application of the test free by employees of the Agricultural Department. This agreement has varied somewhat in its terms during the period the work has been in progress, but all forms have provided for the slaughter of reacting animals under the supervision of federal inspectors. The loss in such cases falls directly on the farmer, since there is no provision for compensation. A copy of the agreement now in use is appended.[a] Since such work was begun, and up to and including June 30, 1909, 2,010 animals have been subjected to primary tests. Of these, 204 have reacted. Of those not reacting to the first test, 1,237 have been retested and 60 reacted. All cattle tested have been duly tagged, in accordance with the terms of the agreements that have been signed.

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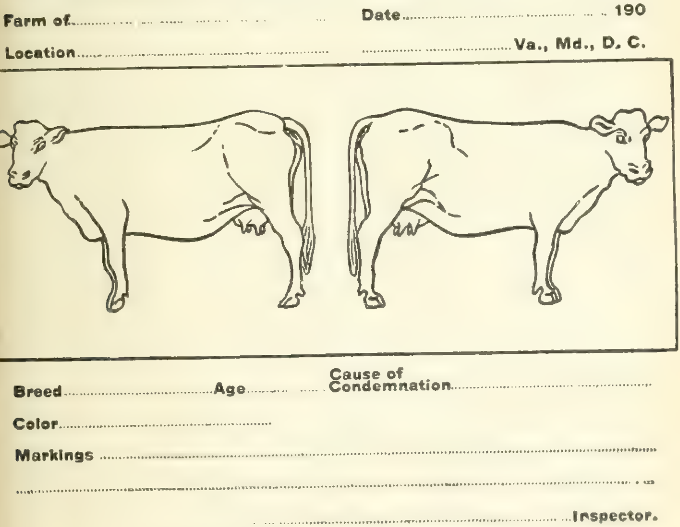
[a] Report of the Health Officer, 1904, page 27.

[b] Hoard's Dairyman, April 5, 1907, page 268.

The identification of tagged cattle is, of course, easy, and therefore animals that have been tuberculin tested are thereafter easily recognized. There is, however, no provision of law that authorizes tagging, and not infrequently difficulty is experienced in identifying and following up cattle condemned by the inspectors of dairy farms merely on the basis of physical examination. Such a cow, if found later on the dairy farm on which it was originally condemned, could, in many cases, be identified without difficulty. But if she had been transferred to any other farm she was less likely to attract attention and even if she did identification was not always easy. To minimize the difficulty of identification, the health department applied to dairy



Farm of.................................... **Date**.................................... 190

Location.................................... ....**Va., Md., D. C.**

Breed....................**Age**.............. **Cause of Condemnation**....................................

Color....................................

Markings....................................

....

....................................**Inspector.**

FIG. 66. Bertillon identification applied to cattle.

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[a] See page 797.

cattle the principle of the Bertillon system of identification. Each inspector is provided with forms giving in profile the figure of a cow, right and left sides, and containing a space for descriptive memoranda. The forms are small, so as to be easily carried in the inspector's pocket. On such a form the inspector notes the characteristic marks of the cow condemned, and the time and place of condemnation. The inspector subsequently carries this form with him for a reasonable time, so that if he finds anywhere a cow that seems to resemble a cow that he has condemned he can confirm or allay his suspicions.

The law regulating the issue of permits to dairy farms outside of the District provides that they shall be issued whenever the health officer is satisfied that the milk from the farm that is about to be licensed will be brought into the District without danger to public health. Since it has been so clearly demonstrated that milk from any herd that has not been tuberculin tested is dangerous to public health, unless effectually pasteurized, the health officer has found it impracticable to issue permits to bring or send milk into the District from such farms. But as the law permits any applicant to bring or send milk into the District from the time he files application and until his application has been rejected, the health officer has, in those cases where the applicant has complied substantially with all requirements of the District laws and regulations, suspended indefinitely action on the application pending the adoption of some general measure looking toward the compulsory tuberculin testing of all cows supplying milk to the District of Columbia or, as an alternative, the compulsory pasteurization of milk from cows not so tested.

Each inspector of dairy farms files with the health officer, weekly, a report of his operations for the preceding week.[a] In addition to this he keeps his own record of outstanding notices, and as soon as practicable after the expiration of the time allowed for the correction of objectionable conditions he visits the premises to see whether the notice has or has not been complied with. If it has been, the inspector makes report accordingly. If it has not, he takes action to enforce compliance. He may immediately serve a notice requiring the licensee to show cause, satisfactory to the health officer, why his permit should not be canceled. Or he may recommend that a letter of that purport be written by the health officer.[b] Or, if the farm be located in the District, he may recommend immediate prosecution in the police court, and with the approval of the health officer he may institute such prosecution. If a licensee has been notified to show cause why his permit should not be canceled, and has failed to do so, or has shown no sufficient cause, then the health officer cancels the permit and notifies the licensee and his consignee or retailer, if he have one, that such action has been taken. If thereafter the milk from that farm is brought into the District, the person at whose instance it is brought is prosecuted in the police court.

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[a] See form for report on page 802.

[b] For copy of form see page 801.

INSPECTION OF DAIRIES.

Two inspectors are available for the inspection of dairies—that is, of places where milk is sold within the District of Columbia—and for the collection of samples of milk. The number of licensed dairies within the district, independent of those located on dairy farms, is 62. The number of places where milk is sold as a mere incident to some other more general business, which places must be regarded as dairies only for purpose of inspection and not for purpose of licensing,[a] is considerable, probably as many as 1,500. The exact number, however, is not known, as such places are registered only as grocery stores, lunch rooms, and so on, and not as milk shops. They begin the sale of milk at the pleasure of the proprietor, discontinue it when he is ready, and resume the business at will; and the health officer knows nothing of it. Many of the latter class of places, however, being grocery stores, come not only under the occasional observation of the inspector of dairies, but also under the more frequent observation of the food inspectors assigned to the supervision of markets and green-grocery stores. In view of the considerable amount of time necessarily consumed in bringing samples of milk collected to the health office from the places of collection, and with a view to increasing the amount of attention paid to the sanitary condition of dairies, it has been deemed best to assign one inspector solely to the sanitary inspection of dairies, requiring him to collect no samples of milk, or to collect them as an incident to his other work. The other inspector is detailed primarily to the collection of samples of milk from dairies, lunch rooms, and grocery stores, and other places where milk is handled for sale, and from the railroad stations where milk is received, any inspections of dairies which he may make being merely incidental thereto. The average number of inspections to which each licensed dairy was subjected during the year ended June 30, 1909, was 20.7. The average number of inspections made daily by the inspector of dairies was 4.3.

In the inspection of dairies, the inspector is guided primarily by the regulations for the government of dairies and dairy farms promulgated under the authority of the act of March 2, 1895. He enforces, however, any and all laws and regulations relating to the sanitary condition of the premises which he visits. Enforcement is ordinarily effected through the service of a notice allowing a certain amount of time for the correction of the objectionable conditions. A carbon copy of each notice is filed with the health officer, with the daily report of the inspector, on the day following the day of service, the original being left with the person to be notified. If the conditions which the inspector finds are so excessively bad as to warrant such action, he not only gives instructions for their immediate correction, but, with the approval of the health officer, institutes criminal proceedings in the police court at once. In the ordinary cases, however, after the expiration of the time allowed by the notice which has been served, the inspector visits the premises and if the objectionable conditions have been corrected he so reports. Otherwise, unless there is reason for allowing further time, police court proceedings are then instituted. A scheme for the rating of dairies has recently been devised and is now in use. A copy of the inspector's score card relating to any given dairy is filed in a jacket reserved for that dairy, with all other papers relating to the establishment, another copy having been furnished the dairyman for his information and guidance.[a]

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[a] See page 762.

INSPECTION OF MILK.

The inspector charged with the collection of samples of milk is expected to bring into the department daily not less than 20 samples of milk. He collected during the year ended June 30, 1909, 3,760 samples of milk and cream, an average of 12.4 samples for each work day, including half holidays as full days. These samples he obtained in the open market from dairies, grocery stores, lunch rooms, or other places where milk is sold, or from milk wagons, or at the railroad stations where milk from the dairy farm first reaches the city. Milk is obtained by purchase whenever anyone is present to receive the money. Money for the purchase of samples is advanced by the inspector out of his own funds, and he is reimbursed each month for the amount thus expended. Vouchers for such reimbursement must be sworn to by the collector and approved by the chemist or the inspector detailed to assist him, such approval being based upon the quantity of milk actually delivered at the laboratory as shown by the laboratory record. The voucher may call for reimbursement for less milk than has been delivered at the laboratory, since in some cases no payment can be made at the time of collection, but it can not call for more.

While one-half pint of milk is sufficient for purposes of analysis, yet in view of a decision of the court of appeals (D. C. *v.* Garrison, 22 Appeals, D. C., 563)[b] it is necessary for the collector to purchase a pint whenever the vendor claims to sell nothing less than pints, unless the inspector is able to show that he sells in smaller quantities. In view of the court's decision, moreover, the department has felt compelled to purchase quart samples whenever it has been alleged by the vendor that he sold only in unbroken packages and had nothing smaller than 1 quart on hand, the department not being prepared to prove a contrary practice. It might be good administration to require the collector to undertake to mix a pint or a quart sample on the premises of the vendor and to take therefrom so much as might be required for analysis and to dispose of the remainder then and there. In view of the difficulty, however, of thoroughly agitating a pint of milk in a pint bottle, or a quart of milk in a quart bottle, and to avoid, in event of prosecution, attack on the ground of the alleged inaccuracy or unfairness of the sample thus taken, it has been deemed best to require the inspector to bring whatever milk he collects to the health office, where the mixing is done in the laboratory. The necessity for carrying such a large amount of milk tends to diminish the working capacity of the inspector. Samples collected are labeled at the time of collection and a record kept by the collector. The samples collected are delivered to the chemist or to his assistant and are immediately analyzed. If the result of the analysis shows that the circumstances warrant such action, prosecution is instituted by the analyst. The inspector who collected the samples testifies as to its origin and the analyst testifies as to its composition.

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[a] For copies of forms used in the dairy-inspection service, see page 806.

[b] For a statement relative to this decision, see page 766.

Arrangements were once made whereby the vendor could, when a sample was purchased, be supplied by the inspector with a portion of it, duly sealed, so that the vendor could have an analysis made independem of the official analysis, if he so desired. This, however, did not do away with the necessity for reserving in the health office a portion of any sample upon an analysis of which prosecution is to be based, since the reserving of such samples is required by statute.

The results of all analyses are transcribed from the laboratory notebook to the official laboratory record, and thereafter are entered upon index cards so that the results of the analyses of all samples procured from any one dealer can be seen by a glance at his laboratory card. On this card are subsequently entered memoranda showing the results of such prosecutions, if any, as are instituted. If the analysis shows that the sample of milk purchased does not conform to the legal standard, the vendor is notified of that fact, and if it is the purpose of the department to institute a prosecution against him, a portion of the sample is reserved, duly sealed and kept under lock and key, so that the vendor may obtain it and submit it to an independent analysis if he so desires. All samples that are found to be of standard quality are delivered to one of the local charitable institutions supported at public expense. It was formerly the custom of the department to inform the vendor of every sample of milk purchased as to the result of the analysis, without reference to the quality of the milk analyzed, whether above or below standard. It was discovered, however, that in some cases, vendors of milk were exhibiting in their places of business official reports of the analysis of samples of high grade, while failing to display reports showing the collection of bad samples. As the practice was liable to mislead the public, the sending out of notices giving information as to the analysis of samples at or above the legal standard was omitted.

No analyses of samples of milk submitted by dealers are made, and only under exceptional circumstances are analyses made of samples of milk submitted by private citizens. It is the general practice of the department with respect to the latter class of samples to make analyses only when the person submitting the samples signifies his ability to testify that the sample submitted is in the same condition which it was when it was left at his residence, and his desire or willingness to prosecute the vendor if the sample be found to be of an unlawful character. Upon receipt, however, of complaint as to the quality of the milk delivered to any individual, whether a consumer or a dealer in milk, the department will, if other official business permits and the complaint seems to justify such action, collect a sample or samples from the suspected vendor through the department's own agents and make the necessary analyses. Examinations of this character for dealers in milk are, however, restricted to the smallest possible number, since if any other course were adopted the requests from dealers for such service would probably be so numerous as to interfere with the general work of the department.

The practice of the department with respect to the analysis of samples of milk for dealers works no hardship on the larger dealer, since for his own protection from a purely commercial standpoint he is or should be prepared to have all milk which he purchases analyzed in his own place of business. The smaller dealer can not well do this, and to him it would be of advantage if from time to time he could obtain analyses of the milk which he purchases, without cost or at a reasonable charge. It would be advisable, therefore, to increase the laboratory facilities of the health department, and to authorize the analysis of samples of milk and of other foods for reasonable fees, so that dealers in milk or of other articles of food or of drugs might be able better to supervise and control the articles which they handle. Fees from such a laboratory might be adjusted so as to make it self-sustaining and to permit the force of the laboratory to be increased, if necessary, in proportion as the work and, therefore, the fees increased. By this method the needs of the dealer could be met and the regular inspection work of the department need not be interfered with.

In order to reduce to a minimum the time spent in the police court by the chemist and his assistant, and by the inspector detailed for the collection of samples of milk, and in order to limit the amount of clerical work connected with the service, cases are not referred daily to the corporation counsel for prosecution, but only at the end of each week. The chemist after the close of the week prepares a report showing the work done in the chemical laboratory. He reports from time to time the names and addresses of all persons who have sold samples of milk or other foods, which have been found to be of an unlawful character. On such reports the chemist recommends either that a prosecution be instituted or that it be not instituted. The minimum amount of butter fat which milk must contain in order to permit its lawful sale as whole milk is 3.5 per cent. This standard is a reasonable one and should insure the sale of a high-grade article. At times, however, even the most careful dealer may allow his milk to fall below it. The practice of the department requires that wherever a sample of milk is found to contain added water or a preservative, or to be colored, or to contain less than 3.25 per cent butter fat, prosecution is to be instituted as a matter of course. If the butter fat is 3.25 per cent or more, but less than 3.5 per cent, then prosecution is or is not instituted according as the entire recent record of the vendor is good or bad. If his milk has been repeatedly below 3.5 per cent, then even though the present sample shows more than 3.25 per cent, prosecution is instituted. In cases within this class, where the element of judgment enters, the inspector submits with his report and recommendations a statement showing the recent record of each vendor. A similar practice is in force with respect to cream, the legal standard for butter fat being 20 per cent, and prosecution being instituted as a matter of course if the amount contained in a given sample falls below 18 per cent, and being instituted or not, according to the entire recent record of the vendor, where the amount of butter fat is 18 per cent or more and yet less than 20 per cent.

In the chemical laboratory are analyzed samples of water from wells on dairy farms. These samples are collected by inspectors of dairy farms, and if from near-by farms are brought to the chemist by the inspector. If from outlying farms, as, for instance, those in the Frederick district, samples are forwarded by express.

CONTAGIOUS-DISEASE SERVICE.

Attention has already been called to the difficulty which an inspector of dairy farms incurs in any effort that he may make to detect on the farm cases of communicable diseases, such as typhoid, scarlet fever, or diphtheria. For the information of those of the readers of this report who are not familiar with technical matters relating to milk inspection, it is necessary to add that by no known method of chemical or bacteriological analysis can the possibility, or even the probability, of the presence in milk of the typhoid bacillus or the diphtheria bacillus be excluded with any reasonable degree of certainty; that the colon bacillus is not an infrequent inhabitant of milk, its presence indicating merely contamination with the excrement of the cow and not even suggesting sewage pollution; and that the organism that causes scarlet fever is as yet entirely unknown. Under such circumstances, the following method has been adopted to facilitate the detection of contagious diseases on the dairy farm:

Upon receipt by the health department of a report of a case of typhoid fever, scarlet fever, or diphtheria, an inspector from the health department visits the premises where the patient is and ascertains the name of the dairyman who furnished the milk consumed by the patient prior to the onset of his illness. The dairyman is immediately notified to discontinue leaving milk bottles at the infected premises or apartment until after the recovery or removal of the patient, of which event he is notified at the time of its occurrence. Bottles containing milk, if left at infected places, may be taken into the sick room and possibly even directly used by the patient, and then, in view of imperfect methods of disinfection adopted by the attendants and of imperfect methods of cleaning adopted by the dairymen, may be the means of spreading disease. The name of the dairyman who supplies milk to the patient having been ascertained, and the dairyman having been notified to discontinue the delivery of bottles at the infected place, the case is entered on the records of the health department to the dairyman's account. This is done by the inspector in charge of the contagious-disease service personally, and if at any time it seems that the number of cases being charged to any one dairyman is out of proportion to the size of his business, due regard being paid to the general extent to which the disease is prevailing in the District, then inquiry is immediately begun to ascertain whether his milk supply is or is not exposed to infection. Such investigations are made by medical officers in the contagious-disease service and by the veterinary surgeons in the milk-inspection service. The local establishment of the milk dealer is visited and an effort made to ascertain whether he or anyone in his family, or any employee or his family, or anyone to whom they have been exposed, is suffering from the disease under consideration. A similar course is pursued with reference to the dairy farm. The water from the dairy farm is analyzed, if there is any indication for such procedure. If the circumstances show that the milk supply is exposed to infection, then immediate action is taken to remove the danger. Permits are suspended or revoked, with or without notice, if circumstances indicate that such action is called for, and are renewed or reissued only after all danger is passed. In some cases it has been found possible to remove the source of danger without putting the dairyman out of business, and whenever possible such measures, less radical so far as the dairyman is concerned, have been adopted. While the method outlined above for detecting and removing danger from infected milk is not ideal, in that it does not reveal the presence or the location of the disease until a small or possibly a large number of persons have been infected, yet, so far as is known, it is the only practicable method that has yet been devised. The safety of the community with respect to the possible spread of communicable diseases of human beings through milk depends primarily on clean intelligent dairying, and for this there is no substitute.

COST OF MILK INSPECTION.

It is not difficult to state exactly the amount appropriated specifically for the milk-inspection service, but it is impossible to do more than to approximate its actual cost. Some of the time of the health officer and of certain other officers and employees in the employ of the health department is given up in part to the service named, and a part of the contingent expenses of the service are so closely bound up with the general contingent expenses that accurate separation is impossible. The following statement shows, however, approximately the present cost of the service per annum:

Annual cost of milk-inspection service.

SPECIFIC APPROPRIATIONS.

1 inspector, dairy farms	$1,200
5 inspectors, dairy farms, at $1,000	5,000
1 inspector, dairies	900
1 inspector, collecting samples	900
1 inspector, assisting chemist	900

GENERAL APPROPRIATIONS.

[Pro rata chargeable to milk-inspection service.]

20 per cent salary of health officer	$800
20 per cent salary of assistant health officer	500
Salary of supervising inspector	1,200
10 per cent salary of chief inspector	180
25 per cent salary of chemist	450
Salary of inspector detailed to assist chemist	900
5 per cent salary of inspector in charge of contagious-disease service	120
20 per cent salary of clerical force of health department	1,760
2 per cent general contingent allotment to health department	70
20 per cent allotment for postage	124
40 per cent contingent fund for chemical laboratory	400
75 per cent appropriation for traveling expenses	4,500
Total	19,904

Statements showing the amount specifically appropriated for the milk-inspection service, and the work accomplished, are appended.[a]

Time and space will not permit a discussion of the claim made by some that the efforts of the Government to bring about an improvement in the milk supply have resulted in an increase in the cost of milk to the consumer. The increase in the price of milk is in keeping with the increase in the price of almost everything else and is territorially too widely spread to have been brought about simply by the enactment or proposed enactment of laws for the improvement of the milk supply. As a matter of fact, too, very few producers of milk in this vicinity have any accurate idea of the actual cost of production or of the net increase in the cost, if any, brought about by the enforcement of existing dairy regulations. Not knowing the cost of production, the individual producer can not fix intelligently the lowest price at which milk must be sold in order to produce a fair profit, but is guided by general impressions only and by prevailing custom. He does undoubtedly know, however, that the cost of production has been increased by higher prices for foodstuffs and for labor; his monthly expenditures must show this. If he proposes to increase the price of milk merely in proportion to the increase in the cost of production and to the increase in the cost of the farmer's living, the public should not complain. But the increase should be fairly and frankly stated, and the necessity for it should not be used unfairly as a club with which to beat down future legislation for the improvement of the milk supply.

RESULTS.

The results of the milk-inspection service must not be measured by bacterial counts or chemical analyses. These are mere incidents. The purpose of the service is to prevent sickness and to save human lives, and by its efficiency in accomplishing these ends it must be judged. In the first place, then, the milk-inspection service has assisted the health department in discovering outbreaks of typhoid fever and scarlet fever, due to milk infection. Of these outbreaks, seven were of typhoid fever and two of scarlet fever.[b] And the milk-inspection service alone has, after the discovery of such outbreaks, enabled the health department usually to locate the very focus of infection, and commonly to do so in time to take effective action to cut short the progress of the disease.

While the relation between the milk supply and the spread of the diseases named above is important, it is less so than is the relation between the milk supply and infant mortality; the death rate of infants is the commonly accepted standard by which the efficiency of the milk-inspection service of any community is measured. It may be claimed, however, and with some show of propriety, that many factors other than improvement in the milk supply have been at work to reduce the number of infantile deaths; or that a diminishing birth rate may account for the lessening of the infantile death rate, computed as that death rate perforce is, upon the total population and not upon the basis of the infantile population alone. To eliminate as nearly as possible error from these causes, no effort has been made to gauge the results of milk inspection by the general infantile mortality, but consideration has been limited to one single class of diseases, the intimate relation between which and the milk supply is almost universally conceded; that is, to diarrhea and enteritis occurring among children under 2 years of age.

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[a] See page 792.

[b] For details as to these outbreaks, see page 51 et seq.

When a sudden drop in the death rate from any particular cause is practically coincident with the inauguration of measures intended to bring about that very result, when there is no other discoverable cause for such drop, and when the lower death rate persists with the continuance of such measures and continues to fall in proportion, more or less, to their efficiency, it is reasonable to suppose that the relation of cause and effect exists. And such are the circumstances with respect to the improvement in the milk supply of the District of Columbia and the diminution in the mortality from diarrheal diseases among persons less than 2 years old. The beginning of a persistent fall in the general death rate appears when we compare the figures for 1892 with those for 1893. A fall in the death rate of infants under 1 year of age appears at the same time. But no permanent lowering appears in the death rate from diarrhea and enteritis among children under 2 years of age until the second year after the enactment of the milk law. This law was enacted March 2, 1895, and a certain period elapsed before it could be put into effective operation. The death rate from diarrhea and enteritis among infants during the fiscal year following its enactment was 168 per 100,000. The next year it had fallen to 151, the third year to 136, and the fourth year to 110. There have, of course, been slight fluctuations. In the calendar year 1900 the death rate rose to 132 per 100,000, but the annual average for the five-year period, 1900-1904, was only 109, and during 1903 it fell to 91. In 1905 the rate was 101, and in 1906 it was 97. In 1907 it was 98, and during 1908 it remained at the same figure. The death rate from diarrhea and enteritis among children under 2 years old during the five-year period preceding the enactment of the milk law, in 1895, was 175 per 100,000. If the same rate had continued during the thirteen years that elapsed after its enactment and prior to December 31, 1908, the number of deaths from these causes would have been approximately 6,949, or 2,386 more than actually occurred. And if the number of fatal cases of diarrhea and enteritis, whether averted through the operation of the milk law or otherwise, was 2,386, how much more numerous must

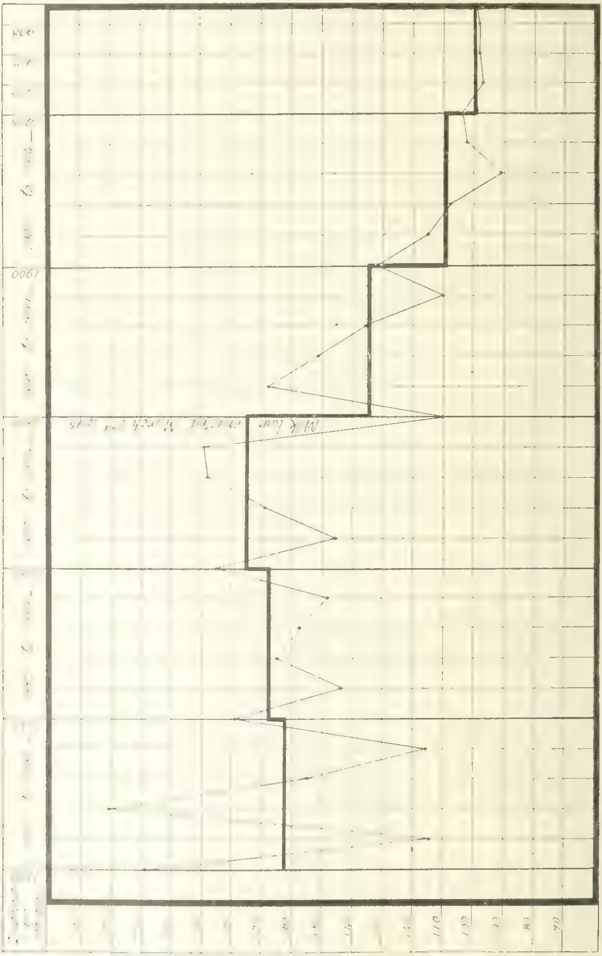

FIG. 67.—Chart showing fall in mortality from diarrhea and enteritis among children under 2 years of age in the District of Columbia subsequent to enactment of milk law in 1895.

have been the cases in which infants were spared attacks of a milder character?

Possibly some of the apparent saving of the lives of 184 infants yearly, and the apparent prevention of sickness, may have been due to a diminishing birth rate, but the records of the health department show no reason for believing that it was all due to that cause. Some of it may have been due to the improvement in the general sanitary condition of the city; some to a better understanding on the parts of parents as to how to care for their children; and some to increasing ability on the part of the medical profession to treat such diseases. These factors, however, had been operating for a long period before the enactment of the milk law, but without apparent effect. The death rate from diarrheal diseases of infants during the five-year period, 1880 to 1884, was 162 per 100,000; during the next period it was 168, and from 1890 to 1894 it was 175. Is there any reason to believe that in 1895, the very year the milk law was enacted, some circumstance, as yet undiscovered, rendered potent these theretofore inert factors, so that in the period from 1895 to 1899 they made the death rate from infantile diarrhea and infantile enteritis fall to 135, during the next period fall to 109, during the year 1905 fall to 101, during the year 1906 to 97, and during 1908 and 1909 to 98? Or was it not the enactment of the milk law in 1895 and the continuous and increasingly efficient enforcement of it that has wrought this result? The facts are here stated, and pending a further study of the matter the reader must be left to draw his own conclusions. This, however, can be said without fear of successful contradiction, that if the enactment of the milk law of 1895 has prevented only one iota of the deaths and the sickness that it seems to have prevented, the milk-inspection service has amply justified its existence.

SUPPLEMENTARY MEMORANDUM—GOVERNMENT OF THE DISTRICT OF COLUMBIA.

In order that readers of this report who do not reside in the District of Columbia may better comprehend the situation that exists there with respect to the supervision of the milk supply, the following statement is made. Those who are residents of the District of Columbia, or most of them, are probably already familiar with everything that it contains.

The District of Columbia covers only 60 square miles of land, lying on the Potomac River between the States of Maryland and Virginia. According to the federal census of 1900 it had a population of 278,718. The police census of 1906 showed, however, a population of 326,435, which is manifestly larger as compared with the federal returns, but is in harmony with police censuses of other recent years. Approximately 30 per cent of the population is colored. In view of its large population and relatively small area, the greater part of the District is urban in character, and most of the milk sup ply is produced in neighboring States. There is in law no delimitation between the present city of Washington and the District of Columbia.

The District of Columbia is not self governing, but is under the control of the Government of the United States. All legislation of any considerable importance is enacted by the Federal Congress, and all appropriations whatsoever are made by it. For the latter purpose all local revenues are paid into the Federal Treasury. As a very general rule, any appropriation that is made comes one-half from the revenues of the District of Columbia and one-half from the revenues of the United States, but appropriations have occasionally been made wholly from local revenues.

For purposes of administration, the immediate direction of the affairs of the District is intrusted to a Board of Commissioners, two of whom are appointed by the President from among the residents of the District of Columbia, and confirmed by the Senate, the third Commissioner being detailed from the Engineer Corps of the Army. The general duties of the Board of Commissioners are executive, but the Board now has a considerable legislative power relating to matters not deemed of such vital importance as to be reserved for the exclusive jurisdiction of Congress.

Laws and regulations relating to public health are executed and enforced by a health officer, appointed by the Commissioners and responsible to them. There has been no board of health in the District of Columbia since 1878, when the present form of government was established. Prior to that date, and as far back as 1871, there had been a board of health created by act of Congress having jurisdiction over the entire District of Columbia, and before that the city of Washington and the city of Georgetown, each then a separate municipal corporation, had their own boards.

EXHIBIT A. *Showing certain death rates in the District of Columbia before and after the enactment of the milk law of March 2, 1895.*

Year.	Death rates per 1,000 of entire population.[a] General death rate.	Of persons 1 year and over.	Of persons under 1 year.	From diarrhea and enteritis under 2 years.
Fiscal year:				
1880	23.88	16.93	6.95	2.09
1881	24.60	18.06	6.54	1.14
1882	22.33	16.47	5.86	2.21
1883	23.74	17.83	5.91	1.55
1884	24.64	18.28	6.36	1.16
Average 5 years	23.85	17.54	6.31	1.62
1885	24.77	18.64	6.13	1.79
1886	22.97	17.03	5.94	1.44
1887	22.30	16.14	6.16	1.65
1888	24.23	17.31	6.92	1.58
1889	23.15	17.28	5.87	1.49
Average 5 years	23.48	17.27	6.21	1.68
1890	23.81	17.43	6.38	1.85
1891	25.16	19.04	6.12	1.46
1892	25.36	18.58	6.78	1.69
1893	23.25	16.96	6.29	1.88
1894	21.89	16.19	5.70	1.89
Average 5 years	23.95	17.70	6.25	1.75
1895[b]	21.23	16.00	5.23	1.10
1896	21.16	15.84	5.32	1.68
1897	19.75	15.05	4.70	1.51
1898	20.54	16.13	4.41	1.36
1899	20.32	15.93	4.39	1.10
Average 5 years	20.59	15.77	4.82	1.35
Calendar year:				
1900	20.61	16.04	4.57	1.32
1901	20.19	16.07	4.12	1.15
1902	18.95	14.91	4.04	1.08
1903	19.08	15.57	3.51	.91
1904	19.61	16.05	3.56	1.02
Average 5 years	19.68	15.73	3.95	1.09
1905	19.20	15.68	3.52	1.04
1906	19.35	15.57	3.78	.97
1907	19.25	15.85	3.40	.98
1908	18.08	14.84	3.24	.98

[a] Reasonably accurate data are not available for the calculation of these infantile death rates upon the basis of the infantile population alone.

[b] The act now regulating the sale of milk in the District was approved March 2, 1895.

EXHIBIT B.—*Cost and work of the milk-inspection service of the District of Columbia*

[Appropriations made for the supervision and control of the milk supply of the District of Columbia.]

Fiscal year.	Number of employees of each grade.							Traveling expenses, outside District of Columbia.	Contingent expenses, chemical laboratory.	Date of appropriation act.
	$400	$900.	$1,000.	$1,200.	$1,500.	$1,600.	$1,800.			
1895-6	[a] 1			[b] 1					$250	Mar. 2, 1895
1896-7				[a] [c] 1	[b] 1					June 11, 1896
1897-8				[a] [c] 1	[b] 1					Mar. 3, 1897
1898-9				[a] [c] 1	[b] 1					June 30, 1898
1899-1900				[c] 1	[b] 1					Mar. 3, 1899
1900-1		1		[c] 1		[b] 1				June 6, 1900
1901-2		5		[c] 1		[b] 1				Mar. 1, 1901
1902-3		7		[c] 1			[b] 1	$1,000	[d] 1,000	Mar. 3, 1903
1903-4		7		[c] 1			[b] 1	1,200		July 1, 1902
1904-5		3	4	[c] 1			[b] 1	1,200 300	[d] 1,000	Apr. 27, 1904 [e] Mar. 3, 1905
1905-6		3	4	[c] 1			[b] 1	1,200	[d] 1,000	Do.
1906-7		3	4	[c] 1			[b] 1	1,500	[d] 1,000	June 27, 1906
1907-8		3	5	[c] 1			[b] 1	2,000	[d] 1,000	Mar. 2, 1907
1908-9		3	5	[c] 1			[b] 1	3,000	[d] 1,000	May 26, 1908

[a] Veterinarian to all branches of the District government.

[b] Chemist; performs all chemical work for the health department and much for other branches of District government.

[c] Inspector of live-stock and dairy farms.

[d] This fund covers all expenses of chemical laboratory, whether related to milk or not.

[e] Deficiency.

[The records of the inspection of dairies has not been kept in such a manner as to permit the making of a statement showing the relative amount of work done from year to year.]

Year.	Number of dairy farms inspected.	Number of inspections of dairy farms.	Number of cattle on dairy farms.	Number of inspections of cattle.	Number of samples of milk analyzed.	Number of samples of cream analyzed
1894-5					545	21
1895-6					479	
1896-7		405			244	6
1897-8		(a)			350	7
1898-9		596		3,240	273	53
1899-1900		806		3,508	413	39
1900-1	841	957		15,459	776	59
1901-2	(b)	2,265		38,645	4,737	334
1902-3	788	3,399	15,930	64,879	6,090	146
1903-4	867	4,092	17,733	69,108	7,798	150
1904-5	930	3,633	16,166	61,708	7,803	427
1905-6	918	3,526	16,250	59,851	6,066	651
1906-7	883	3,932	15,950	65,600	4,511	449
1907-8	904	4,407	16,172	73,142	4,652	381
1908-9	902	1,294	16,116	70,231	3,659	101

[a] The record for the year 1898 fails to show the number of inspections of dairy farms actually made.

[b] The record for the year 1902 fails to show the number of farms actually inspected.

Exhibit C.—*Selected forms used in the milk-inspection service of the District of Columbia.*

APPLICATION FOR PERMISSION TO BRING OR SEND MILK INTO THE DISTRICT OF COLUMBIA.

INSTRUCTIONS TO APPLICANTS.

1. The act to regulate the sale of milk in the District of Columbia provides that no person shall bring or send into the District of Columbia for sale any milk without a permit so to do from the health officer of said District, and that such permits shall be issued subject to the following conditions:

That none but pure and unadulterated milk shall be, with knowledge of its impurity, brought into said District.

That in the management of the dairy farm upon which the milk is produced, or of the dairy at which the milk is collected and stored prior to shipment, the applicant shall be governed by the regulations of the health office of the District of Columbia, approved by the Commissioners of said District, issued for dairies and dairy farms in said District, when said regulations do not conflict with the laws of the State in which said dairy or dairy farm is located.

A copy of the regulations above referred to is herewith inclosed.

The said dairy or dairy farm may be inspected at any time without notice by the health officer of the District of Columbia or his duly appointed representatives.

2. This application should be made out in ink and signed with your *full* name. If applicant is a married woman she should sign her own name in full and insert her husband's name in the space provided for that purpose.

3. To insure the freedom of your dairy herd from tuberculosis it is advised that it be tuberculin tested and suitable disposition made of reacting animals. In no other way can its freedom from tuberculosis be demonstrated. If you desire to have the tuberculin test applied sign the accompanying blank form of agreement for that purpose and return it to this department. It will then be transmitted to the Bureau of Animal Industry, and your herd will be tested without cost to you. This test should by all means be made, thereby removing a source of infection to your well cattle and protecting the persons who consume your milk.

4. Under the law no permit can be issued until after the health officer is satisfied that the milk from the dairy farm to which the permit relates will be brought into the District of Columbia for sale and distribution without danger to public health. Immediately after the filing of the application, however, if it be in proper form, the applicant can lawfully bring or send milk into the District of Columbia and can continue to do so until after his application has been acted upon by the health officer. An inspection of your premises will be made as soon as practicable after the filing of your application.

5. Application for a permit, and the acceptance of a permit, each binds the applicant and licensee to compliance with the conditions stated above. Violation of said conditions renders the permit voidable and unless satisfactorily explained will result in the cancellation of the permit on the books of the health department.

Wm. C. Woodward, M. D.,
Health Officer.

To the Health Officer, District of Columbia:

In compliance with "An act to regulate the sale of milk in the District of Columbia, and for other purposes," I hereby make application for a permit to send or bring milk into said District from the premises described below, located

[Give name of road, nearest cross road, and anything that will aid the inspector in locating premises.]

Number of shipments per day Total number of gallons {Whole milk. Skim milk. Cream.}

Shipped in Wagon ________ Boat Railroad

Time of delivery Place of delivery

Consigned to____

DESCRIPTION OF PREMISES.

STABLE.

Location:

How far from the stable are the hogpens?__________

How far from the stable is the manure stored?___________________________

Is there any standing water near the stable?_____________________________

Is the stable used for any other than dairy purposes?_____________________

Room for cattle:

Size: Feet long____________feet wide_________average height, feet________

Total clear air space per stall, in cubic feet____________________________

Floor:

Kind: Cement____________________ Plank______________________________

How drained?____

What becomes of the drainage?____

Lighting and ventilation:

Number of windows: Glass ____ Muslin curtain____

Total number of square feet in glass windows___________________________

Total number of square feet in glass per stall_____________________________

Total number of square feet in muslin windows__________________________

Total number of square feet in muslin windows per stall__________________

Number of adjustable windows____ Number of nonadjustable windows___

Ventilation other than by windows:

Number of inlets__________________ Total area in square feet___________

Number of outlets_________________ Total area in square feet____________

Location of inlets____

Location of outlets____

Stalls:

How many?_________ Size of each, feet long_________, feet wide_________

What kind of tie is used for the cattle?__________________________________

Is bedding used for the cattle?_____ Kind______________________________

Feed mangers:

Plank Cement____

If a continuous trough in front of the line of stalls, is each animal allotted space separated by a partition?

How are feed troughs cleaned?____

How are cattle watered?

From well-------------spring-------------running stream-------------

If from well, state location, distance, and slope of ground from

Nearest privy--

Hogpens--

Stable--

Barnyard--

Give the approximate depth of well------------feet--------

How is well protected against surface drainage?----------

Has the water any perceptible odor?------ -color?-------taste?-------

BARNYARD.

Size: Feet long-------------------feet wide----------------------------

What disposition is made of the drainage from the barnyard?-------------

Is manure stored in the barnyard?--------------If so, is it protected by a barrier to prevent cattle from walking through it?---------------------

If manure is not stored in barnyard, what disposition is made of it?-------

DAIRY ROOM.

Size. Feet long---------------------------feet wide------------------------

Construction of. Floor-------------Walls------------Ceiling------------

How is dairy room drained?--

Location.

How far is dairy room located from stable?-----------------------------

How far is dairy room located from privy?------------------------------

How far is dairy room located from manure pile?------------------------

How far is dairy room located from hogpens or any other source of contamination?--

Is the dairy room effectually screened?--------------------------------

Do you have a separate wash room for dairy utensils?-------------------

If so, describe the same---

METHODS OF HANDLING MILK.

Kind of receptacles used?---

How are the dairy utensils cleaned?-------------------------------

How is water heated for cleaning dairy utensils?------------------

How much water can be heated at one time?-------------------------

Are dairy utensils sterilized with steam or boiling water?-----------

Are the dairy utensils aired in sunlight after cleaning?------------

Is the milk cooled immediately after milking?-----------------------

Where is the milk cooled?

How is the milk cooled?

To what temperature is the milk cooled?---------------------------

Are cans of milk or cream iced during transportation in hot weather?

Are they covered with wet blanket?

Do you use small top milking pail?

Source of water supply for washing dairy utensils--

If from well, state location, distance, and slope of ground from

Nearest privy ____________

Hogpens ____________

Stable ____________

Barnyard ____________

Give the approximate depth of well____________feet.

How is well protected against surface drainage?____________

Has the water any perceptible odor?______ color?______ taste?______

PRIVY.

Location ____________

Is the excreta deposited directly on ground or in a receptacle?____________

If receptacle is used, is it water-tight?____________

How often are the contents of the receptacle removed and where are they deposited?____________

Are covers provided for the seats?______ Is privy effectually screened?______

CATTLE.

How many milch cows are usually kept?____________

Have the dairy cattle been tuberculin tested?____________

How are cows cleaned?____________ How often____________

Do milkers wash and wipe the udders before milking?____________

Do milkers wash their hands before milking?____________

What facilities are provided, if any, in the stable or dairy, or both, for cleansing the hands of those persons who work with or about the cattle or handle milk or the dairy utensils?____________

Signature of Applicant____________

Post-Office Address____________

Husband's Name____________

[To be filled in if applicant is a married woman.]

Veterinarian's Certificate.

[The veterinarian's certificate must be from one who has regularly graduated from a veterinary medical college, or who practices under a license from a state examining board. If unable to secure the services of such, so state on your application blank, and, all other conditions being satisfactory, action will be suspended until this requirement is met, thus enabling you to send or bring milk into the District without violation of existing law.]

Sir: I have carefully examined the cattle upon the premises above referred to, and their condition is as follows: ____________

Signature____________

Address____________

Personally appeared before me this —— day of ———, 19 , the subscriber, who being duly sworn deposes and says that he is a veterinary surgeon practicing in accordance with the laws of the State in which he resides, and that he has personally examined the cattle referred to in the above statement and knows them to be the same as are referred to in the application to which the certificate is appended, and that their condition is correctly described without evasion or concealment.

Signature____________

Address____________

UNITED STATES DEPARTMENT OF AGRICULTURE.

Bureau of Animal Industry.

AGREEMENT.

In consideration of the testing of my herd of cattle by the Bureau of Animal Industry of the United States Department of Agriculture, and the assistance of said bureau in enabling me to produce and maintain a herd of cattle free from the contamination of tuberculosis, I, ________________________________

[Name of owner.]

of ____, owner of said herd of cattle,

[Post-office address.]

comprising ____.

[Number and kinds over 6 months old; number and kinds under 6 months old.]

do hereby agree as follows:

1. I will cause all animals which react to the tuberculin test, and which also show other marked symptoms of tuberculosis, to be slaughtered within a reasonable time under the United States meat-inspection regulations, and I will cause the carcasses of said animals to be disposed of according to the meat-inspection regulations of the Bureau of Animal Industry, based upon the lesions found upon inspection.

2. I will cause all animals which react to the tuberculin test, but which show no other evidence of tuberculosis, either to be slaughtered and disposed of as herein provided for animals which show also other evidence of tuberculosis, or I will cause such animals to be removed from the herd and portion of the farm upon which the healthy animals of the herd are maintained, and I will cause the diseased animals to be segregated from the healthy animals, and thereafter they shall remain so segregated.

3. In all cases where the milk from such segregated reacting cows is to be used for any purpose whatever I will cause the said milk to be sterilized.[a]

4. Segregated reacting bulls may be used for breeding, provided they are held on leash and are not permitted to leave the premises reserved for their use, and provided the healthy cows bred to such bulls are not unduly exposed to infected premises or to other diseased cattle.

5. I will cause the young from segregated reacting animals to be removed from their mothers at birth, and will not permit the said young to suck their mothers.

6. Any of my premises contaminated by reacting animals will be submitted by me to a thorough disinfection under the direction or supervision of the Bureau of Animal Industry.

7. All cattle owned by me, both healthy and tuberculous, I will mark, or allow to be marked, in such manner as to enable their identity to be retained, and I will not change the location of or slaughter any tuberculosis cattle except after due and timely notification to the Bureau of Animal Industry, stating the exact nature of the change of location, or the exact date, name, and address of the official establishment at which the animal or animals are to be slaughtered.

8. I will add no cattle to the said herd which have not passed a tuberculin test with the tuberculin prepared by the Bureau of Animal Industry, admin-

---

[a] An act to regulate the sale of milk in the District of Columbia states that it shall not be lawful for any person or persons to sell or offer for sale, within the District of Columbia, milk taken from any cow which is known to be suffering from tuberculosis.

istered by a licensed veterinarian of the State, an authorized public agent qualified to perform such test, or by an inspector of the Bureau of Animal Industry: *Provided*, That I may purchase cattle to be added to my herd if the said cattle are kept effectually separated from the rest of my tuberculin tested cattle until the same can be tuberculin tested, and I will immediately notify the Bureau of Animal Industry and the local board of health that these cattle are on my premises subject to test.

9. I will comply with all reasonable sanitary measures which are indicated by the proper officials of the State or Territory wherein my herd is located, or by the local board of health under whose permit I am disposing of dairy products, or by the Bureau of Animal Industry.

In witness whereof I have signed this agreement this --------------- day of -------------------, one thousand nine hundred and ------

Owner of the ---- herd.

(Address) ----

Witness:

APPLICATION FOR PERMISSION TO MAINTAIN A DAIRY WITHIN THE DISTRICT OF COLUMBIA.

INSTRUCTIONS TO APPLICANTS.

1. The act to regulate the sale of milk in the District of Columbia provides that "no person shall maintain a dairy within the District of Columbia without a permit so to do from the health officer of said District."

2. This application should be made out in ink and signed with your *full* name.

3. Under the law no permit can be issued until after the dairy to which this permit relates is found to conform to the regulations governing dairies and dairy farms within the District of Columbia. Immediately after the filing of the application, however, if it be in proper form, the applicant can lawfully begin business and continue the same until after his application has been acted upon by the health officer. An inspection of your premises will be made as soon as practicable after the filing of your application.

4. A copy of the regulations above referred to is inclosed herewith.

WM. C. WOODWARD, M. D.,
Health Officer.

To the Health Officer, District of Columbia.

SIR: In compliance with "An act to regulate the sale of milk in the District of Columbia, and for other purposes," I hereby make application for a permit to maintain a dairy, described below, located------------------------------

[Give street and number.]

DESCRIPTION OF PREMISES

Construction of building {Wood.
Brick.

Number and location of rooms used for dairy purposes____

Ventilation and light. Number of windows ____; doors ____

Floor: Wood, concrete, brick, brick and concrete____

How is the floor drained?____

State location of water-closets, privies, and urinals on the premises____

Are premises used for any purpose other than the sale of dairy products? If so, what?____

Water for cleaning {Steam
Hot.
Cold.

Describe facilities for cleaning cans, bottles, and utensils____

Describe facilities for storing____

How is milk cooled?____

Is the dairy screened as required by law?____

Gallons sold daily {Milk.
Cream.
Buttermilk.

Number of receptacles used____; kind____

Number of wagons____

List of shippers____

If business is to be operated under a trade name, please state such name

____

Respectfully,

Name ____

Address ____

ACKNOWLEDGMENT OF RECEIPT OF APPLICATIONS.

COMMISSIONERS OF THE DISTRICT OF COLUMBIA,
HEALTH DEPARTMENT,
DAIRY AND DAIRY FARM INSPECTION SERVICE,
Washington, D. C., ____, *19*__.

This department is in receipt of your application for

permission to {conduct a dairy in____
conduct a dairy farm in
bring or send milk and cream into} the District of Columbia

Having filed the said application you may now lawfully begin business and continue the same until final action has been taken upon your application. An inspection of your establishment will be made at an early date.

Respectfully,

____, M. D.,
Health Officer.

Please preserve this card as evidence of your authority to engage in the above business.

HEALTH DEPARTMENT, DISTRICT OF COLUMBIA.

No .

DAIRY PERMIT.

Permission is hereby granted to maintain a dairy at subject to regulations governing dairies within the District of Columbia

____________, M. D.,
Health Officer.

, 190

Issued in accordance with an "Act to regulate the sale of milk in the District of Columbia, and for other purposes," approved March 2, 1895.

This permit is not transferable, and applies only to the premises specified hereon. If location is changed, new permit is required.

HEALTH DEPARTMENT, DISTRICT OF COLUMBIA.

No

DAIRY FARM PERMIT.

Permission is hereby granted______to maintain a dairy farm at______subject to regulations governing dairy farms within the District of Columbia.

____________, M. D.,
Health Officer.

, 190__

Issued in accordance with an "Act to regulate the sale of milk within the District of Columbia, and for other purposes," approved March 2, 1895.

This permit is not transferable.

HEALTH DEPARTMENT, DISTRICT OF COLUMBIA.

No .

MILK IMPORTER'S PERMIT.

Permission is hereby granted to bring or send milk into the District of Columbia from the dairy farm located at and described in application No. , subject to the following conditions:

That none but pure and unadulterated milk shall be, with knowledge of its impurity, brought into said District.

That in the management of the dairy farm upon which the milk is produced, or at the dairy at which the milk is collected and stored prior to shipment, the applicant shall be governed by the regulations of the health office of the District of Columbia, approved by the Commissioners of said District, issued for dairies and dairy farms in said District, when said regulations do not conflict with the law of the State in which said dairy or dairy farm is located.

That said dairy or dairy farm may be inspected at any time without notice by the health officer of the District of Columbia or his duly appointed representative.

, M. D.,
Health Officer.

,190

Issued in accordance with an "Act to regulate the sale of milk in the District of Columbia, and for other purposes," approved March 2, 1895.

This permit is not transferable.

Notice of violation of dairy regulations.

Any objection to this notice should be filed with the health officer before the expiration of the time allowed for making the changes specified.

HEALTH DEPARTMENT, DISTRICT OF COLUMBIA.

No. ------

DAIRY AND DAIRY FARM INSPECTION.

Washington, --- *190*--

Mr. -------

SIR: Your attention is called to the following violations of the Regulations for the Government of Dairies and Dairy Farms, which have been found to exist upon your premises:

------ -- - ---- - - -- --- ----

You are hereby notified to correct the same within -- days from the date of service of this notice.

By order of the health officer.

----------,

Inspector of Dairies and Dairy Farms.

Cancellation of permit—preliminary inspector's notice.

HEALTH DEPARTMENT OF THE DISTRICT OF COLUMBIA.

DAIRY AND DAIRY FARM INSPECTION.

, *190* .

Mr. ----------

SIR: An inspection of your dairy farm this date shows that you are violating the conditions under which your permit, No. ------, was issued, namely, ------.

You are therefore directed to show cause in writing to the health officer on or before------, 190--, why your permit should not be canceled.

By order of health officer.

---- - ,

Inspector.

Cancellation of permit—health officer's preliminary letter.

Washington, , *190*

Mr. -

SIR: I have the honor to inform you that the report of Inspector ------, of this department, dated------, 190--, shows that you are violating the conditions under which your permit, No. ------, to bring or send milk into the District of Columbia was issued, namely, . In view of this fact, you are hereby directed to show cause on or before why your permit should not be revoked.

Respectfully,

, M. D.,

Health Officer.

Inspector.

Sir: [illegible]

HEALTH DEPARTMENT OF THE DISTRICT OF COLUMBIA.

Weekly report of inspection of dairy farms, dairies, and live stock.

[Insert remarks, if necessary, on other side of this sheet.]

Week ending ....

		Dairy farms.											Dairies.			Live-stock inspections.									Contagious diseases investigated.					
		Farms inspected.			Cows.											Inspected.				Condemned.										
						Condemned.				Notices served.																				
Day.	Name of owner.	District of Columbia.	Maryland.	Virginia.	Inspected.	Tuberculosis.	Diseased udder.	Other causes.	Total.	Condemned cattle.	Other causes.	Sample of water.	Inspected.	Notices served.	Slaughterhouses visited.	Steers.	Sheep.	Calves.	Hogs.	Steers.	Sheep.	Calves.	Hogs.	Notices served.	Rabies.	Glanders.	Typhoid.		Traveling expenses.	Special reports.
Monday.																														
Tuesday.																														
Wednesday.																														
Thursday.																														
Friday.																														
Saturday.																														
	Total....																													

HEALTH DEPARTMENT OF THE DISTRICT OF COLUMBIA,

Dairy and Dairy-Farm Inspection Service.

SCORE CARD FOR DAIRY FARMS.

Farm of ______ Location ______ Consignee ______

Permit number ______ D. C., Md., Va. ______ 19__ Rating ______

Equipment.	Score. Perfect.	Score. Allowed.
Cows, 16.		
Comfort:		
Bedding	1	
Temperature of stable	1	
Food	2	
Water:		
Clean	1	
Fresh	1	
Light: Four square feet or more of glass per cow (Three square feet, 3; 2 square feet, 2; 1 square foot, 1.)	4	
Ventilation: Automatic system. (Adjustable windows, 1.)	3	
Cubic feet air space per cow, 600 to 1,000 feet (Less than 600 feet, 2; less than 500 feet, 0.)	3	
Stables, 6.		
Location of stable:		
Well drained	1	
Free from contaminating surroundings	1	
Construction of stable:		
Tight, sound floor and proper gutter	2	
Smooth, tight walls and ceiling	1	
Proper stall, tie, and manger	1	
Utensils, 14.		
Construction of utensils	1	
Water for cleaning: Clean, convenient, and sufficient	2	
Facilities for steam (Hot water sufficient to immerse utensils, 2.)	4	
Small-top milking pail	3	
Milk cooler	2	
Clean milking suits	2	
Handling the milk, 4.		
Location of milk room:		
Free from contaminating surroundings	1	
Convenient	1	
Construction of milk room:		
Floors, walls, and ceiling	1	
Light, ventilation, and screens	1	
Total	40	

Methods.	Score. Perfect.	Score. Allowed.
Cows and stables, 16.		
Cleanliness of stables:		
Floor	2	
Walls	1	
Ceiling and ledges	1	
Mangers and partitions	1	
Windows	1	
Stable air	6	
Barnyard: Clean and well drained	2	
Removal of manure daily to field or pit (Manure stored less than 50 feet from stable, 0.)	2	
Utensils and milking, 24.		
Care and cleanliness of utensils:		
Thoroughly cleansed	6	
Steaming or scalding utensils	6	
Inverting utensils in pure air and sunlight	3	
Cleanliness of milking:		
Clean, dry hands	3	
Udders washed and dried. (Udders cleaned with moist cloth, 4.)	6	
Handling the milk, 20.		
Cleanliness of attendants	2	
Milk of each cow removed immediately from the stable	2	
Cleanliness of milk room	3	
Prompt cooling (cooled immediately after milking each cow)	2	
Efficient cooling: Below 50° F. (51° to 55°, 4; 56° to 60°, 2.)	5	
Storage: Below 50° F. (51° to 55°, 2; 56° to 60°, 1.)	3	
Transportation: Iced (For jacket or wet blanket allow 2; dry blanket or covered wagon, 1.)	3	
Total	60	

Score for equipment ______ + Score for method ______ = Final score ______

Source of water supply ______ General condition of farm ______

Violations of regulations, sec. ______ Violations of act, sec. ______

Notices served, to correct, to show cause by ______

Remarks ______

______ *Inspector.*

Score for cattle.

[Printed on back of score card for dairy farm.]

Number cattle in dairy herd	Perfect score	Total possible score for herd.
	For each cow or bull, 100.	

Deductions on account of cattle diseased, etc.

Number of cattle.	Nature of disease, defect, etc.	Deductions per cow.	Total deductions.
	1. Tuberculosis as shown by a physical examination, or by the tuberculin test.	100	
	2. Absence of a tuberculin test within one year of the date of inspection *not to include cattle scored under paragraph 1*	30	
	3. Inflammatory diseases of the udder	100 or less	
	4. Diseases other than or in addition to the diseases mentioned above.	100 or less	
	5. Unclean condition of the teats and udders	40 or less	
	6. Unclean condition of the cows other than specified in the preceding paragraph	30 or less	
	7. Undue emaciation or cows otherwise out of condition	10 or less	

Total deduction for herd ....

Net score .....

Net score ( ) divided by the total possible score for herd ( ) equals percentage score

Remarks

Inspector.

The health department believes that if a cow is suffering from tuberculosis, her entire value as a dairy cow is gone. If she is suffering from an inflammatory disease of the udder, as well as from tuberculosis, she becomes even a greater danger to the herd. And if she is furthermore otherwise diseased, or out of condition, or dirty, she becomes even a more serious menace to public health. For these reasons the above system of scoring has been arranged so that an individual cow may count against the score of the entire herd more than would have been allotted to her had she been in perfect condition.

All cows stabled with the dairy herd or found in the milking line will be scored as part of the herd.

HEALTH DEPARTMENT OF THE DISTRICT OF COLUMBIA.

Dairy and Dairy Farm Service.

SCORE CARD FOR DAIRIES.

Owner or manager ______ Trade name ______

Street and No.______ Permit No ________

Number of wagons ______

Gallons sold daily { Milk ______ / Cream ______ / Buttermilk ______

Date of inspection ______ 190_

Equipment	Score. Perfect.	Score. Allowed.
Plant:		
Location... 10		
Convenience	2	
Surroundings	8	
Arrangement ... 5		
Proper rooms	4	
Convenience	1	
Construction 10		
Floor and drainage	6	
Walls	3	
Ceiling	1	
Light	5	
Ventilation	5	
Screens	5	
Machinery and utensils .30 (Kind, quality, condition, and arrangement.)		
Bottle and can washer	5	
Bottling machine	5	
Capping machine	5	
Crates, racks, etc	5	
Cold storage	5	
Pasteurizer	5	
Water for cleaning ... 20		
Steam	10	
Hot water	5	
Cold water	5	
Salesroom 10		
Location	3	
Construction	4	
Equipment	3	
	100	
Additional deductions for exceptionally bad conditions.		
Total deduction		
Net total		

Methods.	Score. Perfect.	Score. Allowed.
Plant:		
Cleanliness 15		
Floor	3	
Walls	2	
Ceilings	1	
Doors	1	
Windows	1	
Good order	1	
Free from odors	1	
Freedom from flies	5	
Machinery and utensils—		
Cleanliness	30	
Milk—(Handling) 20		
Clarifying	5	
Bottling	10	
Pasteurizing	5	
Storage	20	
45° F. or below... 20		
45° to 50° F 15		
50° to 55° F 10		
Salesroom—		
Cleanliness	10	
Attendants' cleanliness	5	
	100	
Additional deductions for exceptionally bad conditions.		
Total deductions		
Net total		

Score for equipment ______ multiplied by 1__

Score for methods ______ multiplied by 2__

Total to be divided by 3

Final score ______

Supplemental score for wagons.	Perfect.	Allowed.
Construction	3	
Condition	7	
	10	

This score does not cover quality of milk purchased by this dealer.

______

Inspector.

Collection of samples. Inspector's memorandum and label.

I. S. No. .. , 190. Sold as. .. ..

Label...

..

....

Bought by dealer of...

Dealer........ ....

....

Salesman........... Price paid ....per..............

Bought by............... on..... ...at..... ..{a. m. / p. m.

Delivered to............. on... ..at.........{a. m. / p. m.

Remarks.........................

....

....

Inspector.

BUREAU OF CHEMISTRY.

I. S. No.

BUREAU OF CHEMISTRY.

I. S. No.

BUREAU OF CHEMISTRY.

I. S. No.

....

Inspector's description of sample.

HEALTH DEPARTMENT OF THE DISTRICT OF COLUMBIA.
Washington, ... , *190*..

REPORT OF ANALYSIS.

Substances offered for sale or sold as
Milk Cream Skimmed milk
by
at on

Analysis.

	1	2	3	4	5	6	7	8	9	10
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Fat										
Solids not fat										
Water										
*Odor										
*Acidity										
*Added coloring matter										
*Preservative										

*A check indicates that no test was made for the ingredient or quality checked.

Remarks:

, *Analyst.*

Legal standards.—Milk.—Not less than 3½ per cent fat, 9 per cent solids not fat, and not more than 87½ per cent water.
Cream.—Not less than 20 per cent fat.
Skimmed milk.—Not less than $9\frac{3}{10}$ per cent solids, including fat.

Wholesome milk must come from healthy cows living under proper sanitary conditions. It must have been properly cared for at the time of milking and continually thereafter; especially must it have been kept cold. This report shows the chemical composition of the milk analyzed, but indicates only in an imperfect manner its wholesomeness, which can be determined only by considering the condition of the cows, dairy farm, and dairy in connection with this analysis.

There is as yet no fixed standard for acidity in milk, but any sample of milk or cream found to be, in the judgment of the health department, too acid will be regarded as unwholesome, and the seller prosecuted accordingly. The sale of milk, cream, or skimmed milk which has been colored or to which a preservative of any sort has been added is in violation of law.

Dealers in foodstuffs, including, of course, milk, etc., are required by law to know at all times the quality of all goods which they sell. Prosecutions will therefore be instituted in suitable cases without notice.

________________________, M. D.,
Health Officer.

To ______________________

EXHIBIT D.

Laws and regulations relating to the production and sale of milk in the District of Columbia.

ACTS OF CONGRESS.

AN ACT To regulate the sale of milk in the District of Columbia, and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, That from and after the passage of this act no person shall, within the District of Columbia, keep or maintain a dairy or dairy farm without a permit so to do from the health officer of said District; application for said permit shall be made in writing, upon a form prescribed by said health officer: *Provided,* That no applicant for said permit shall be restrained from conducting business until said application has been acted upon by the health officer of the District of Columbia or his duly appointed agent. It shall be the duty of said health officer, upon receipt of said application in due form, to make or cause to be made an examination of the premises which it is intended to use in the maintenance of said dairy or dairy farm; if after such examination said premises are found to conform to the regulations governing dairies and dairy farms within the District of Columbia, said health officer shall issue the permit hereinbefore specified, without charge: *Provided,* That said permit may be suspended or revoked at any time, without notice, by said health officer whenever the milk supply from said dairy or dairy farm is exposed to infection by Asiatic cholera, anthrax, diphtheria, erysipelas, scarlet fever, smallpox, splenic fever, tuberculosis, typhoid fever, typhus fever, or yellow fever, so as to render its distribution dangerous to public health.

SEC. 2. That no person shall bring or send into the District of Columbia for sale any milk without a permit so to do from the health officer of said District; application for said permit shall be made in writing, upon a form prescribed by said health officer, and shall be accompanied by such detailed description of the dairy farm or dairy where said milk is produced or stored as said health officer may require, and by a sworn statement as to the physical condition of the cattle supplying said milk: *Provided,* That no applicant for said permit shall be restrained from conducting business until said application has been acted upon by the health officer of the District of Columbia or his duly appointed agent. If after examination of said application said health officer is satisfied that said milk will be brought into the District of Columbia for sale or consumption without danger to public health, he shall issue, without charge to the applicant, a permit so to do, on condition that none but pure and unadulterated milk shall be, with knowledge of its impurity, brought into said District; that in the management of said dairy or dairy farm said applicant shall be governed by the regulations of the health office of the District of Columbia, approved by the Commissioners of the District of Columbia, issued for dairies and dairy farms in said District, when said regulations do not conflict with the law of the State in which said dairy or dairy farm is located, and that said dairy or dairy farm may be inspected at any time without notice by the health officer of the District of Columbia or his duly appointed representative: *Provided*, That said permit may be suspended or revoked at any time without notice by said health officer whenever the milk supply from said dairy or dairy farm is exposed to infection by Asiatic cholera, anthrax, diphtheria, erysipelas, scarlet fever, smallpox, splenic fever, tuberculosis, typhoid fever, typhus fever, or yellow fever, so as to render its distribution dangerous to public health.

SEC. 3. That no person suffering from, or who has knowingly, within a period specified by the health officer of the District of Columbia, been exposed to diphtheria, scarlet fever, erysipelas, smallpox, anthrax, or other dangerous contagious disease, shall work or assist in or about any dairy or dairy farm; no proprietor, manager, or superintendent of any dairy or dairy farm within the District of Columbia shall knowingly permit any person suffering, or exposed as aforesaid, to work or assist in or about said dairy or dairy farm.

SEC. 4. That all milk wagons shall have the name of the owner, the number of permit, and the location of dairy from which said wagons haul milk, painted thereon plainly and legibly.

SEC. 5. That all grocers, bakers, and other persons having or offering for sale milk shall at all times keep the name or names of the dairymen from whom the milk on sale shall have been obtained posted up in a conspicuous place wherever such milk may be sold or kept for sale.

SEC. 6. That no person shall offer or have for sale in the District of Columbia any unwholesome, watered, or adulterated milk, or milk known as swill milk, or milk from cows that are fed on swill, garbage, or other like substance, nor any butter or cheese made from any such milk.

SEC. 7. (Repealed by act of February 17, 1898. See Wiegand *v.* D. C., 31 Wash. Law Rep., 730.)

SEC. 8. That no person shall sell, exchange, or deliver, or have in his custody or possession with intent to sell, exchange, or deliver, skimmed milk containing less than nine and three-tenths per cent of milk solids, inclusive of fat.

SEC. 9. That no dealer in milk, and no servant or agent of such a dealer, shall sell, exchange, or deliver, or have in his custody or possession with intent to sell, exchange, or deliver, milk from which the cream, or any part thereof, has been removed, unless in a conspicuous place, above the center or upon the outside of every vessel, can, or package thereof, in which milk is sold, the words "skimmed milk" are distinctly marked in gothic letters, not less than one inch in length.

SEC. 10. That it shall not be lawful for any person or persons to sell or offer for sale, within the District of Columbia, milk taken from any cow less than fifteen days before or ten days after parturition, or from any cow which is known to be suffering from tuberculosis, splenic fever, anthrax, or any general or local disease which is liable to render the milk from said cow unwholesome.

SEC. 11. That it shall be the duty of the health officer of the District of Columbia, under direction of the Commissioners of said District, to make and enforce regulations to secure proper water supply, drainage, ventilation, air space, floor space, and cleaning of all dairies and dairy farms within said District; to secure the isolation of cattle suffering from any contagious disease, and to carry into effect the provisions of this act.

SEC. 12. That the health officer of the District of Columbia, or his duly appointed assistants, shall have the right to enter, without previous notice, for the purpose of inspection, any dairy or dairy farm within said District.

Sec. 13. (Repealed by act of February 17, 1898. See Weigand *v.* D. C., 31 Wash. Law Rep., 730.)

Sec. 14. That prosecutions under this act shall be in the police court of said District, on information signed by the attorney of the District or one of his assistants, and any person or persons violating any of the provisions of this act shall be deemed guilty of a misdemeanor, and shall, on conviction, be punished for the first offense by a fine of not less than five dollars nor more than twenty-five dollars, to be collected as other fines and penalties, or by imprisonment in the workhouse for a period of not more than thirty days, and for the second offense and each subsequent offense, by a fine of not less than fifty dollars nor more than one hundred dollars, or by imprisonment in the workhouse for ninety days, or by both such fine and imprisonment, in the discretion of the court, and if the person so convicted of a second or subsequent offense hold a permit under this act, the same shall be canceled and no permit shall be issued to said person for a period of six months: *Provided*, That any person or persons under this act shall have the privilege, when demanded, of a trial by jury as in other jury cases in the police court.

Sec. 15. That all laws and parts of laws inconsistent with the foregoing be, and the same are hereby, repealed.

Approved, March 2, 1895.

AN ACT Relating to the adulteration of foods and drugs in the District of Columbia.

[30 Stats., 246.]

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, That no person shall, within the District of Columbia, by himself or by his servant or agent, or as the servant or agent of any other person, sell, exchange, or deliver, or have in his custody or possession with the intent to sell or exchange, or expose or offer for sale or exchange, any article of food or drug which is adulterated within the meaning of this act.

Sec. 2. That the term "drug," as used in this act, shall include all medicines for external or internal use, antiseptics, disinfectants, and cosmetics. The term "food," as used herein, shall include confectionery, condiments, and all articles used for food or drink by man, and if there be more than one quality of any article of food or drug known by the same name the best quality thereof shall be furnished to the purchaser, unless he otherwise requests at the time of making such purchase, or unless he be notified at such time of the inferior quality of the article delivered.

Sec. 3. That an article shall be deemed to be adulterated within the meaning of this act:

(a) In the case of drugs: First, if, when sold under or by a name recognized in the United States Pharmacopœia, it differs from the standard of strength, quality, or purity laid down in the edition thereof at the time official; second, if, when sold under or by a name not recognized in the United States Pharmacopœia, but which is found in the German, French, or English Pharmacopœia, it differs from the strength, quality, or purity laid down therein; third, if, when sold as a patented medicine, compounded drug, or mixture it is not composed of all the ingredients advertised or printed or written on the bottles, wrappers, or labels of or on or with the patented medicine, compounded drug, or mixture: *Provided*, That if the defendant in any prosecution under this act, in respect to the sale of any such patented medicine, compounded drug, or mixture, shall prove to the satisfaction of the court that he had purchased the article in question as the same in nature, substance, and quality as that demanded of him by the purchaser, and with a written warranty to that effect; that he had no reason to believe at the time when he sold it that the article was otherwise, and that he sold it in the same state as when he purchased it, he shall be discharged from the prosecution.

(b) In the case of food: First, if any substance or substances have been mixed with it so as to reduce or lower or injuriously affect its quality or strength; second, if an inferior or cheaper substance or substances have been substituted wholly or in part for it; third, if any valuable constitutent has been wholly or in part abstracted from it; fourth, if it is an imitation of or is sold under the name of another article; fifth, if it consist wholly or in part of a deceased, decomposed, putrid, or rotten animal or vegetable substance, whether manufactured or not; sixth, if it is colored, coated, polished, or powdered whereby damage is concealed, or if it is made to appear better or of greater value than it really is; seventh, if it contains any added poisonous ingredient or any ingredient which may render it injurious to the health of a person consuming it; eighth, in the case of milk, if it contains less than three and one-half per centum of fat, less than nine per centum of solids not fat, and contains more than eighty-seven and one-half per centum of water; in the case of cream, if it contains less than twenty per centum of butter fat; ninth, in the case of butter or cheese, if it is not made exclusively from milk or cream, or both, with or without common salt; the butter, if it contains more than twelve per centum of water, more than five per centum of salt, and less than eighty-three per centum of fat; tenth, in the case of coffee, if it is not composed entirely of the seed of the Caffea arabica; eleventh, in the case of lard, if it is not made exclusively from the rendered fat of the healthy hog; twelfth, in the case of tea, if it is not composed entirely of the genuine leaf of the tea plant not exhausted; thirteenth, in the case of all kinds of vinegar, if it contains an acidity equivalent to the presence of less than four per centum of absolute acetic acid; and cider vinegar, if it is not made from the pure apple juice and contains less than one and five-tenths per centum of total solids; fourteenth, in the case of cider, if it is not made from the legitimate product of pure apple juice; in the case of wines and fruit juices, if not made from the pure fruit as represented; and in the case of cider, wines, fruit juices, and malt liquors, if not free from salicylic acid or other preservatives; and in the case of malt liquors, if not free from picric acid, cocculus indicus, colchicine, colocynth, aloes, and wormwood; fifteenth, in the case of glucose, if it contains more than five one-hundredths per centum of ash; sixteenth, in the case of flour, if it is not composed entirely of one single ground cereal; seventeenth, in the case of bread, if there is any addition of alum, sulphate of copper, borax, or sulphate of zinc, or other poisonous or harmful ingredient, and if it contains more than thirty-one per centum of moisture, more than two per centum of ash, and less than six and twenty-five one-hundredths per centum of albuminoids; eighteenth, in the case of olive oil, if it is not made exclusively from the olive berry (Olea europaea), and its specific gravity at fifteen and six-tenths degrees centigrade; (sixty degrees Fahrenheit) "actual density" to be not more than nine hundred and seventeen one-thousandths nor less than nine hundred and fourteen one-thousandths: *Provided*, That an offense shall not be deemed to be committed under this section in the following cases, that is to say, first, where the order calls for an article of food or drug inferior to such standard, or where such difference is made known by being plainly written or printed on the package; second, where the article of food or drug is mixed with any matter or ingredient not injurious to health and not intended fraudulently to increase its bulk, weight, or measure or conceal its inferior quality, if at the time such article is delivered to the purchaser it is made known to him that such article of food or drug is so mixed.

SEC. 4. That it shall be the duty of the health officer of the District of Columbia, under the direction of the Commissioners of said District, to adopt such measures as may be necessary to facilitate the enforcement hereof, and prepare rules and regulations with regard to the proper method of collecting and examining drugs and articles of food in said District.

SEC. 5. That it shall be the duty of the health officer to investigate a complaint for a violation of any of the provisions of this act on the information of any person who lays before him satisfactory evidence by which to substantiate such complaint.

SEC. 6. That every person offering for sale or delivering to any purchaser any drug or article of food included in the provisions of this act shall furnish to any analyst or other officer or agent of the health department, who shall apply to him for the purpose and shall tender him the value of the same, a sample sufficient for the purpose of analysis of any such drug or article of food which is in his possession.

SEC. 7. That in all cases where any drug or article of food shall be taken as a sample to be examined and analyzed the person making the analysis shall reserve a portion of the sample, which shall be sealed, for a period of thirty days from the time of taking such sample, and in case of a complaint the reserved portion alleged to be adulterated shall, upon application, be delivered to the defendant or his attorney.

SEC. 8. That no person shall hinder, obstruct, or in any way interfere with any inspector, analyst, or other person of the health department in the performance of his duty in carrying out the provisions of this act.

SEC. 9. That all prosecutions under this act shall be in the police court of said District, on information brought in the name of the District of Columbia, and on its behalf; and any person or persons violating any of the provisions of this act shall be deemed guilty of a misdemeanor, and upon conviction shall be punished by a fine of not less than five dollars nor more than one hundred dollars.

SEC. 10. That all acts and parts of acts inconsistent with this act be, and the same are hereby, repealed: *Provided*, That nothing in this act contained shall be construed as modifying or repealing any of the provisions of "An act defining butter, also imposing a tax upon and regulating the manufacture, sale, importation, and exportation of oleomargarine," approved August second, eighteen hundred and eighty six, or of "An act defining cheese, and also imposing a tax upon and regulating the manufacture, sale, importation, and exportation of 'filled cheese,'" approved June sixth, eighteen hundred and ninety-six.

Approved, February 17, 1898.

AN ACT To prevent the adulteration of candy in the District of Columbia

[30 Stat., 398.]

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, That no person or corporation shall, by himself, his servant, or agent, or as the servant or agent of any other person or corporation, manufacture for sale or knowingly sell or offer to sell any candy adulterated by the admixture of terra alba, barytes, talc, or any other mineral substance, by poisonous colors or flavors, or other ingredients deleterious or detrimental to health.

Sec. 2. That any person or corporation convicted of violating any of the provisions of this act shall be punished by a fine not exceeding one hundred dollars. The candy so adulterated shall be forfeited and destroyed under the direction of the court.

Sec. 3. That it is hereby made the duty of the prosecuting attorneys of the District of Columbia to appear for the people and to attend to the prosecution of all complaints under this act in all the courts of said District.

Sec. 4. That this act shall take effect upon its passage.

Approved, May 5, 1898.

AN ACT For preventing the manufacture, sale, or transportation of adulterated or misbranded or poisonous or deleterious foods, drugs, medicines, and liquors, and for regulating traffic therein, and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, That it shall be unlawful for any person to manufacture within any Territory or the District of Columbia any article of food or drug which is adulterated or misbranded, within the meaning of this act; and any person who shall violate any of the provisions of this section shall be guilty of a misdemeanor, and for each offense shall, upon conviction thereof, be fined not to exceed five hundred dollars or shall be sentenced to one year's imprisonment, or both such fine and imprisonment, in the discretion of the court, and for each subsequent offense and conviction thereof shall be fined not less than one thousand dollars or sentenced to one year's imprisonment, or both such fine and imprisonment, in the discretion of the court.

Sec. 2. That the introduction into any State or Territory or the District of Columbia from any other State or Territory or the District of Columbia, or from any foreign country, or shipment to any foreign country of any article of food or drugs which is adulterated or misbranded, within the meaning of this act, is hereby prohibited; and any person who shall ship or deliver for shipment from any State or Territory or the District of Columbia to any other State or Territory or the District of Columbia, or to a foreign country, or who shall receive in any State or Territory or the District of Columbia from any other State or Territory or the District of Columbia, or foreign country, and having so received, shall deliver, in original unbroken packages, for pay or otherwise, or offer to deliver to any other person, any such article so adulterated or misbranded within the meaning of this act, or any person who shall sell or offer for sale in the District of Columbia or the Territories of the United States any such adulterated or misbranded foods or drugs, or export or offer to export the same to any foreign country, shall be guilty of a misdemeanor, and for such offense be fined not exceeding two hundred dollars for the first offense, and upon conviction for each subsequent offense not exceeding three hundred dollars or be imprisoned not exceeding one year, or both, in the discretion of the court: *Provided*, That no article shall be deemed misbranded or adulterated within the provisions of this act when intended for export to any foreign country and prepared or packed according to the specifications or directions of the foreign purchaser when no substance is used in the preparation or packing thereof in conflict with the laws of the foreign country to which said article is intended to be shipped; but if said article shall be in fact sold or offered for sale for domestic use or consumption, then this proviso shall not exempt said article from the operation of any of the other provisions of this act.

Sec. 3. That the Secretary of the Treasury, the Secretary of Agriculture, and the Secretary of Commerce and Labor shall make uniform rules and regulations for carrying out the provisions of this act, including the collection and examination of specimens of foods and drugs manufactured or offered for sale in the District of Columbia, or in any Territory of the United States, or which shall be offered for sale in unbroken packages in any State other than that in which they shall have been respectively manufactured or produced, or which shall be received from any foreign country, or intended for shipment to any foreign country, or which may be submitted for examination by the chief health, food, or drug officer of any State, Territory, or the District of Columbia, or at any domestic or foreign port through which such produce is offered for interstate commerce, or for export or import between the United States and any foreign port or country.

Sec. 4. That the examinations of specimens of foods and drugs shall be made in the Bureau of Chemistry of the Department of Agriculture, or under the direction and supervision of such bureau, for the purpose of determining from such examinations whether such articles are adulterated or misbranded within the meaning of this act; and if it shall appear from any such examination that any of such specimens is adulterated or misbranded within the meaning of this act, the Secretary of Agriculture shall cause notice therof to be given to the party from whom such sample was obtained. Any party so notified shall be given an opportunity to be heard, under such rules and regulations as may be prescribed as aforesaid, and if it appears that any of the provisions of this act have been violated by such party, then the Secretary of Agriculture shall at once certify the facts to the proper United States district attorney, with a copy of the results of the analysis or the examination of such article duly authenticated by the analyst or officer making such examination, under the oath of such officer. After judgment of the court, notice shall be given by publication in such manner as may be prescribed by the rules and regulations aforesaid.

Sec. 5. That it shall be the duty of each district attorney to whom the Secretary of Agriculture shall report any violation of this act, or to whom any health or food or drug officer or agent of any State, Territory, or the District of Columbia shall present satisfactory evidence of any such violation, to cause appropriate proceedings to be commenced and prosecuted in the proper courts of the United States, without delay, for the enforcement of the penalties as in such case herein provided.

Sec. 6. That the term "drug" as used in this act shall include all medicines and preparations recognized in the United States Pharmacopœia or National Formulary for internal or external use, and any substance or mixture of substances intended to be used for the cure, mitigation, or prevention of disease of either man or other animals. The term "food," as used herein, shall include all articles used for food, drink, confectionery, or condiment by man or other animals, whether simple, mixed, or compound.

Sec. 7. That for the purposes of this act an article shall be deemed to be adulterated:

In case of drugs:

First. If, when a drug is sold under or by a name recognized in the United States Pharmacopœia or National Formulary, it differs from the standard of strength, quality, or purity, as determined by the test laid down in the United States Pharmacopœia or National Formulary official at the time of investigation: *Provided*, That no drug defined in the United States Pharmacopœia or National Formulary shall be deemed to be adulterated under this provision if the standard of strength, quality, or purity be plainly stated upon the bottle, box, or other container thereof, although the standard may differ from that determined by the test laid down in the United States Pharmacopœia or National Formulary.

Second. If its strength or purity fall below the professed standard or quality under which it is sold.

In the case of confectionery:

If it contains terra alba, barytes, talc, chrome yellow, or other mineral substance or poisonous color or flavor, or other ingredient deleterious or detrimental to health, or any vinous, malt, or spirituous liquor or compound or narcotic drug.

In the case of food:

First. If any substance has been mixed and packed with it so as to reduce or lower or injuriously affect its quality or strength.

Second. If any substance has been substituted wholly or in part for the article.

Third. If any valuable constituent of the article has been wholly or in part abstracted.

Fourth. If it be mixed, colored, powdered, coated, or stained in a manner whereby damage or inferiority is concealed.

Fifth. If it contain any added poisonous or other added deleterious ingredient which may render such article injurious to health: *Provided*, That when in the preparation of food products for shipment they are preserved by any external application applied in such manner that the preservative is necessarily removed mechanically, or by maceration in water, or otherwise, and directions for the removal of said preservative shall be printed on the covering or the package, the provisions of this act shall be construed as applying only when said products are ready for consumption.

Sixth. If it consists in whole or in part of a filthy, decomposed, or putrid animal or vegetable substance, or any portion of an animal unfit for food, whether manufactured or not, or if it is the product of a diseased animal, or one that has died otherwise than by slaughter.

Sec. 8. That the term "misbranded" as used herein shall apply to all drugs, or articles of food, or articles which enter into the composition of food, the package or label of which shall bear any statement, design, or device regarding such article, or the ingredients or substances contained therein which shall be false or misleading in any particular, and to any food or drug product which is falsely branded as to the State, Territory, or country in which it is manufactured or produced.

That for the purposes of this act an article shall also be deemed to be misbranded:

In case of drugs.

First. If it be an imitation of or offered for sale under the name of another article.

Second. If the contents of the package as originally put up shall have been removed, in whole or in part, and other contents shall have been placed in such package, or if the package fail to bear a statement on the label of the quantity or proportion of any alcohol, morphine, opium, cocaine, heroin, alpha or beta eucaine, chloroform, cannabis indica, chloral hydrate, or acetanilide, or any derivative or preparation of any such substances contained therein.

In the case of food:

First. If it be an imitation of or offered for sale under the distinctive name of another article.

Second. If it be labeled or branded so as to deceive or mislead the purchaser, or purport to be a foreign product when not so, or if the contents of the package as originally put up shall have been removed in whole or in part and other contents shall have been placed in such package, or if it fail to bear a statement on the label of the quantity or proportion of any morphine, opium, cocaine, heroin, alpha or beta eucaine, chloroform, cannabis indica, chloral hydrate, or acetanilide, or any derivative or preparation of any of such substances contained therein.

Third. If in package form, and the contents are stated in terms of weight or measure, they are not plainly and correctly stated on the outside of the package.

Fourth. If the package containing it or its label shall bear any statement, design, or device regarding the ingredients or the substances contained therein, which statement, design, or device shall be false or misleading in any particular: *Provided*, That an article of food which does not contain any added poisonous or deleterious ingredients shall not be deemed to be adulterated or misbranded in the following cases:

First. In the case of mixtures or compounds which may be now or from time to time hereafter known as articles of food, under their own distinctive names, and not an imitation of or offered for sale under the distinctive name of another article, if the name be accompanied on the same label or brand with a statement of the place where said article has been manufactured or produced.

Second. In the case of articles labeled, branded, or tagged so as to plainly indicate that they are compounds, imitations, or blends, and the word "compound," "imitation," or "blend," as the case may be, is plainly stated on the package in which it is offered for sale: *Provided*, That the term blend as used herein shall be construed to mean a mixture of like substances, not excluding harmless coloring or flavoring ingredients used for the purpose of coloring and flavoring only: *And provided further*, That nothing in this act shall be construed as requiring or compelling proprietors or manufacturers of proprietary foods which contain no unwholesome added ingredient to disclose their trade formulas, except in so far as the provisions of this act may require to secure freedom from adulteration or misbranding.

Sec. 9. That no dealer shall be prosecuted under the provisions of this act when he can establish a guaranty signed by the wholesaler, jobber, manufacturer, or other party residing in the United States, from whom he purchases such articles, to the effect that the same is not adulterated or misbranded within the meaning of this act, designating it. Said guaranty, to afford protection, shall contain the name and address of the party or parties making the sale of such articles to such dealer, and in such case said party or parties shall be amenable to the prosecutions, fines, and other penalties which would attach, in due course, to the dealer under the provisions of this act.

Sec. 10. That any article of food, drug, or liquor that is adulterated or misbranded within the meaning of this act, and is being transported from one State, Territory, District, or insular possession to another for sale, or, having been transported, remains unloaded, unsold, or in original unbroken packages, or if it be sold or offered for sale in the District of Columbia or the Territories, or insular possessions of the United States, or if it be imported from a foreign country for sale, or if it is intended for export to a foreign country, shall be liable to be proceeded against in any district court of the United States within the district where the same is found, and seized for confiscation by a process of libel for condemnation. And if such article is condemned as being adulterated or misbranded, or of a poisonous or deleterious character, within the meaning of this act, the same shall be disposed of by destruction or sale, as the said court may direct, and the proceeds thereof, if sold, less the legal costs and charges, shall be paid into the Treasury of the United States, but such goods shall not be sold in any jurisdiction contrary to the provisions of this act or the laws of that jurisdiction: *Provided, however*, That upon the payment of the costs of such libel proceedings and the execution and delivery of a good and sufficient bond to the effect that such articles shall not be sold or otherwise disposed of contrary to the provisions of this act, or the laws of any State, Territory, District, or insular possession, the court may by order direct that such articles be delivered to the owner thereof. The proceedings of such libel cases shall conform, as near as may be, to the proceedings in admiralty, except that either party may demand trial by jury of any issue of fact joined in any such case, and all such proceedings shall be at the suit of and in the name of the United States.

SEC. 11. The Secretary of the Treasury shall deliver to the Secretary of Agriculture, upon his request from time to time, samples of foods and drugs which are being imported into the United States or offered for import, giving notice thereof to the owner or consignee, who may appear before the Secretary of Agriculture, and have the right to introduce testimony, and if it appear from the examination of such samples that any article of food or drug offered to be imported into the United States is adulterated or misbranded within the meaning of this act, or is otherwise dangerous to the health of the people of the United States, or is of a kind forbidden entry into, or forbidden to be sold or restricted in sale in the country in which it is made or from which it is exported, or is otherwise falsely labeled in any respect, the said article shall be refused admission, and the Secretary of the Treasury shall refuse delivery to the consignee and shall cause the destruction of any goods refused delivery which shall not be exported by the consignee within three months from the date of notice of such refusal, under such regulations as the Secretary of the Treasury may prescribe: *Provided*, That the Secretary of the Treasury may deliver to the consignee such goods pending examination and decision in the matter on execution of a penal bond for the amount of the full invoice value of such goods, together with the duty thereon, and on refusal to return such goods for any cause to the custody of the Secretary of the Treasury, when demanded, for the purpose of excluding them from the country, or for any other purpose, said consignee shall forfeit the full amount of the bond: *And provided further*, That all charges for storage, cartage, and labor on goods which are refused admission or delivery shall be paid by the owner or consignee, and in default of such payment shall constitute a lien against any future importation made by such owner or consignee.

SEC. 12. That the term "Territory" as used in this act shall include the insular possessions of the United States. The word "person" as used in this act shall be construed to import both the plural and the singular, as the case demands, and shall include corporations, companies, societies, and associations. When construing and enforcing the provisions of this act, the act, omission, or failure of any officer, agent, or other person acting for or employed by any corporation, company, society, or association, within the scope of his employment or office, shall in every case be also deemed to be the act, omission, or failure of such corporation, company, society, or association as well as that of the person.

SEC. 13. That this act shall be in force and effect from and after the first day of January, nineteen hundred and seven.

Approved, June 30, 1906.

AN ACT To amend section eight hundred and seventy-eight of the Code of Law for the District of Columbia.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, That section eight hundred and seventy-eight of the Code of Law for the District of Columbia be, and the same is hereby, amended by adding thereto the following:

"Sec. 878a. That the following words shall, in addition to their ordinary meaning, have the meaning herein given: The word 'person' or 'persons,' in sections eight hundred and seventy-eight b, c, d, e, and g, inclusive, shall include 'firms' or 'corporations;' the word 'vessel' or 'vessels' in sections eight hundred and seventy-eight b, c, d, and e, shall include 'cans,' 'bottles,' 'siphons,' and 'boxes;' the word 'mark' or 'marks' shall include 'labels,' 'trade-marks,' and all other methods of distinguishing ownership in vessels, whether printed upon labels or blown into bottles or engraved and impressed upon cans or boxes.

"Sec. 878b. That persons engaged in producing, manufacturing, bottling, or selling milk or cream, or any other lawful beverage composed principally of milk, in vessels, with their name, trade-mark, or other distinctive mark, and the word 'registered' branded, engraved, blown, or otherwise produced thereon, or on which a pasted trade-mark label is put upon which the word 'registered' is also distinctly printed, may file with the clerk of the supreme court of the District of Columbia a description by facsimile, or a sample of an original package so marked or branded or blown, showing plainly such names and marks thereon, together with their name in full, or their corporate name, and also their place of business in the District of Columbia, and if so filed shall cause the same to be published for not less than two weeks successively in a daily or weekly newspaper published in the District of Columbia.

"Sec. 878c. That whoever, except the person who shall have filed and published a description of the same as aforesaid, fills with milk or cream, or other beverage, as aforesaid, with intent to sell the same, any vessel so marked and distinguished as aforesaid, the description of which shall have been filed and published as provided in the preceding section, or defaces, erases, covers up, or otherwise removes or conceals any such name or mark as aforesaid, or the word 'registered,' thereon, or sells, buys, gives, takes or otherwise disposes of, or traffics in the same without having purchased the contents thereof from the person whose name is in or upon such vessel, or without the written consent of such person, shall, for the first offense, be punished by a fine of not less than fifty cents for each such vessel, or by imprisonment for not less than ten days nor more than one year, or both such fine and imprisonment; and for each subsequent offense by a fine of not less than one or more than five dollars for each such vessel, or by imprisonment for not less than twenty days nor more than one year, or by both such fine and imprisonment.

"Sec. 878d. That the use or possession by any person not engaged in the production or sale of milk or cream or other beverage as aforesaid, except the person who shall so have filed and published a description of the same as aforesaid, of any vessel marked or distinguished as aforesaid, the description of which shall have been filed and published as aforesaid, without purchase of the contents thereof from, or the written consent of, the person who shall so have filed and published the said description, shall be prima facie evidence of the unlawful use, possession of, or traffic in, such vessel, and the person so using or in possession of the same, except the person who shall so have filed and published the said description as aforesaid, shall be punished as in the next preceding section provided.

"Sec. 878e. That upon complaint of any person who has complied with section eight hundred and seventy-eight b, or his agent, to the police court of the District of Columbia, or one of the judges thereof, that such person, or agent, has reason to believe, and does believe, that any person within the District of Columbia is guilty of the violation of any provision of this act, the said court or judge may issue a search warrant to discover and obtain such vessels as aforesaid and their contents, and may also cause to be brought before the said court or judge the person so believed to be guilty, or his agent or employee, in whose possession or upon whose wagon or premises any such vessel or vessels may be found; and any such person, agent, or employee found guilty of a violation of any of the provisions of this act shall be punished as aforesaid, and the said court or judge shall also order the property taken upon any such search warrant to be delivered to its owner.

"Sec. 878f. That the clerk of the supreme court of the District of Columbia is hereby authorized to make regulations and prescribe forms for the filing of labels, trade-marks, or other distinctive marks under the provisions of the foregoing amendments to section eight hundred and seventy-eight.

"Sec. 878g. That nothing in the foregoing amendments to section eight hundred and seventy-eight shall prevent or restrain any person who is the legal owner of a trade-mark or label from proceeding in an action of tort against any person found guilty of violating any subsection of section eight hundred and seventy-eight."

Approved, February 27, 1907.

Orders of the Commissioners of the District of Columbia.

POLICE REGULATIONS.

Cow yards, pens, or stables.

Article XIX. Section 1. No person shall establish or maintain a cow yard, pen, or stable within any of the more densely populated parts of the District of Columbia, within two hundred feet of any building used as a dwelling house, manufactory, store, or place of public assemblage, without the written consent of the owner of such building; such consent to be renewed upon the first day of July of each year upon thirty days' notice by the health officer to that effect: *Provided*, That nothing in this section shall be construed to prevent a person from keeping one cow for his own domestic use, nor to prevent the sale of the surplus milk by a person keeping a cow for his own domestic use.

Sec. 2. Any person violating any of the provisions of this regulation shall, upon conviction thereof, be punished by a fine of not more than five dollars for each day during which such violation shall continue.—*Order of August 28, 1897.*

BUILDING REGULATIONS.

Location of dairies.

Sec. 170a. No dairy nor establishment for the storage or sale of milk or other dairy products, which shall involve in its use or operation more than two wagons, shall be established or located in any square or block fronting on any street or avenue where more than one-half of the improved property in such square or block fronting on such street or avenue is used for residential purposes, nor shall any such dairy or establishment be located in any square or block fronting on any alley of which more than three-fourths of the improved property in such square or block is used for residential purposes, except with the written consent of the owners of three-fourths of the property within two hundred feet of the boundaries of the site on which such dairy or establishment is proposed to be located: *Provided*, That this regulation shall not apply to any case of rebuilding or enlarging, in the same location, any existing dairy or establishment for the storage or sale of milk or other dairy products.—*Order of April 17, 1906.*

REGULATIONS FOR THE GOVERNMENT OF DAIRIES AND DAIRY FARMS.

Ordered, That the following regulations made by the health officer of the District of Columbia, pursuant to the requirements of section 11 of "An act to regulate the sale of milk in the District of Columbia, and for other purposes," approved March 2, 1895, in lieu of the regulations on the same subject made and approved June 26, 1895, are hereby approved:

SECTION 1. No building or space shall be used for dairy purposes which is not well lighted and ventilated, which is not provided with a suitable floor, and, if such room or space be a cellar or subcellar, or be located in a cellar or subcellar, which is not properly concreted, guttered, and drained.

SEC. 2. No dairy shall be located or maintained within any kitchen, wash room, workshop, or inhabited room, nor in proximity to any water closet, privy, cesspool, or urinal, nor in any room or space which is not of such size and construction as to permit the entire separation of all milk and milk products, both in the process of handling and storing the same, from all probable sources of contamination, either by dirt, noxious gases, infective organisms or substances, or anything liable to alter unnecessarily the quality of such milk or milk products.

SEC. 3. Every person maintaining a dairy shall provide for the use thereof, and shall use, a sufficient number of receptacles, made of nonabsorbent material, for the reception, storage, and delivery of milk, and shall cause them to be kept clean and wholesome at all times; and having delivered any such receptacle to a consumer shall not again use the same for the reception, storage, or delivery of milk or cream in any form until it has been, to his personal knowledge, properly cleaned after such use.

SEC. 3a. Elsewhere than in the proper parts of premises which have been duly constructed and equipped, and which are duly maintained for the handling, storage, and sale of milk or cream, no person shall fill or partly fill with milk or cream any receptacle intended for delivery to a customer unless such receptacle, at the time of filling, be furnished by the customer for whose service such receptacle is intended.—(Amendment of July 14, 1903.)

SEC. 4. Every person maintaining a dairy shall provide for the use thereof a supply of pure and suitable water, sufficient for the proper washing of all cans, bottles, and appliances.

SEC. 5. Every person maintaining a dairy shall keep the same and all appurtenances thereto clean and wholesome at all times, and shall change the water in the coolers at least once each day.

SEC. 6. No building shall be used for stabling cows for dairy purposes which is not well lighted, ventilated, drained, and constructed, or which is not provided with stalls or with proper stanchions for anchoring the cows, so arranged as to allow not less than three and one half feet width of space for each milch cow; or which is not provided with good and sufficient facilities for feeding the animals in a cleanly manner; or which contains less than six hundred cubic feet clear air space for each cow, unless the use of such building for stabling cows for dairy purposes has been authorized prior to the promulgation of these regulations, in which case it shall contain not less than five hundred cubic feet clear air space for each cow.

SEC. 7. No room shall be used for stabling cows for dairy purposes which contains any water-closet, privy, cesspool, urinal, or manure pit, nor shall any fowl, hog, horse, sheep, or goat be kept in any room used therefor.

SEC. 8. Every person using any premises for keeping cows for dairy purposes shall, when so directed by the health officer, erect and maintain in the stable, stall, shed, or yard connected therewith one or more proper receptables for drinking water for such cows, and shall keep the same supplied with clean, fresh water, and none other.

SEC. 9. Every person using any premises for keeping cows for dairy purposes shall keep the entire premises clean and in good repair and the buildings well painted or whitewashed.

SEC. 10. Every person using any premises for keeping cows for dairy purposes shall cause the dung to be removed from the stables at least twice daily, and always within one hour preceding every milking of the cows; and shall not allow any accumulation of dung within the building occupied by the cows, but shall, whenever in the opinion of the health officer it is required by local conditions and surroundings, provide temporary storage for the same and for other refuse in a separate place, which shall be covered, and which, when so ordered by said health officer, shall be a water-tight receptacle.

SEC. 11. Every person keeping cows for dairy purposes within the city of Washington or its more densely populated suburbs, or elsewhere in the District of Columbia, if, in the opinion of the health officer, local conditions require it, shall cause the inclosure in which such cows are kept to be graded and drained so as to keep the surface reasonably dry and to prevent the accumulation of water therein, except as may be permitted for the purpose of supplying drinking water; and shall not permit any garbage, urine, fecal matter, or similar substance to be placed or to remain in such inclosure, nor any open drain to run through it.

SEC. 12. Every person keeping cows for the production of milk for sale shall cause them to be kept clean and wholesome at all times, and shall cause the teats and, if necessary, the udder to be carefully cleaned by brushing, washing, or wiping before milking, and shall cause each such cow to be properly fed and watered.

SEC. 13. Any person using any premises for keeping cows for dairy purposes shall provide and use a sufficient number of receptacles, of nonabsorbent material, for the reception, storage, and delivery of milk, and shall keep them clean and wholesome at all times, and at milking time shall remove each receptacle, as soon as filled, from the stable or room in which the cows are kept; nor shall any milk or cream be stored or kept within any room used for stabling cows or other domestic animals.

SEC. 14. It shall be the duty of every person having charge or control of any premises upon which cows are kept to notify the health officer of the District of Columbia of the existence of any contagious or infectious disease among such cows, by letter delivered or mailed, within twenty-four hours after the discovery thereof, and to thoroughly isolate any cow or cows so diseased, or which may reasonably be believed to be infected, and to exercise such other precautions as may be directed in writing, by said health officer.

SEC. 15. Milkers and those engaged in the handling of milk or cream shall maintain strict cleanliness of their hands and persons while milking or while so engaged. It shall be the duty of every person holding a permit to maintain a dairy or dairy farm to enforce this regulation in reference to such persons as may assist them in the maintenance thereof.

SEC. 16. That any person violating any of the foregoing regulations shall, on conviction thereof in police court, be punished by a fine of not more than ten dollars for each and every such offense, to be collected as other fines and penalties are collected.

SEC. 17. That the regulations for the government of dairies and dairy farms in the District of Columbia promulgated June 26, 1895, are hereby repealed.—*Order of July 31, 1897.*

AN ORDINANCE To prevent the sale of unwholesome food in the cities of Washington and Georgetown, as amended by Commissioners' orders May 15, 1871

[Except as otherwise indicated these ordinances were promulgated by the board of health May 15, 1871, and have been legalized by Congress on April 24, 1880, and again on August 17, 1894.]

SEC. 2. That no person shall offer for sale within the District of Columbia any liquor used for drink, whether malt, vinous, or ardent, or the milk of cows or goats, intended to be used for food or drink, which has been adulterated with any poisonous or deleterious ingredient; and any person violating the provisions of this section shall, upon conviction, be punished by a fine of not less than ten nor more than fifty dollars for each and every such offense.

SEC. 5. That no person, whether owner, manager, keeper of, agent, bartender, or clerk, in any saloon, restaurant, boarding house, or eating house, located within the District of Columbia, shall offer for sale as food or drink anything poisonous or unwholesome; and any person violating the provisions of this section shall, upon conviction thereof, be punished by a fine of not less than five nor more than twenty-five dollars for each and every such offense.

SEC. 6a. That any person in the District of Columbia who receives milk or cream for sale shall, immediately after emptying the receptacle in which such milk or cream has been received, thoroughly rinse such receptacle so as to free the same from all remnants of milk and of cream, or shall cause such receptacle to be so rinsed; and no person in said District shall put or, having power and authority to prevent, permit to be put into any receptacle which is commonly used for the storage or delivery of milk or cream for sale anything which is filthy or offensive or any refuse matter of any kind. Any person violating the provisions of this section shall, upon conviction thereof, be punished by a fine not exceeding twenty-five dollars for each and every such offense.—*Commissioners' regulation of April 21, 1903.*

SEC. 6b. That no occupant of any building, room, stand, stall, or other place in the District of Columbia where cattle, sheep, hogs, poultry, or other animals are slaughtered or killed, and no occupant of any building, room, stand, stall, or other place in said District where milk, game, poultry, fish, vegetables, fruits, groceries, or other articles of food are prepared, kept, sold, or offered for sale, shall permit such place or an appurtenance thereto to be unnecessarily unclean and unwholesome. No person who slaughters or kills in said District any cattle, sheep, hogs, poultry, or other animals, and no person who prepares, keeps, sells, or offers for sale any meat, game, poultry, fish, vegetables, fruits, groceries or other article of food, shall permit any implement, knife, measure, or utensil used in connection therewith to be unnecessarily unclean or unwholesome or in unfit condition for use in connection with the slaughtering or killing of cattle, sheep, hogs, poultry, or other animals, or for the preparation, keeping, selling, offering for sale, and delivery of meat, game, poultry, fish, vegetables, fruits, groceries, or other articles of food.

Any person who violates any of the provisions of this regulation shall, upon conviction thereof, be punished by a fine of not more than twenty-five dollars for each and every offense.—*Commissioners' Regulation of October 6, 1904.*

Sec. 6e. No person shall expose for sale on any public highway, or in any uninclosed market, store, shop, stand, or stall, or on any open lot, or transport over any public highway for sale either by himself or by any other person, in the District of Columbia, any meat, fish, plucked poultry or game bird, dressed rabbit or squirrel, butter, butterine, oleomargarine, lard, lard compound or substitute, cheese, candy, cake, bread, dates, figs, or any food whatsoever of a kind not commonly washed, peeled, shelled, or cooked before being eaten, unless the same be effectually and in a cleanly manner wrapped, or covered and inclosed, so as to protect it from dust and insects.

No person shall expose for sale in any place aforesaid between April 1 and October 31, inclusive, of any year, any fresh meat or fresh fish unless said meat or fish, while thus exposed, be kept at a temperature not exceeding fifty-five degrees Fahrenheit.—*Commissioners' Regulation of May 11, 1909.*

Sec. 6f. No vendor or distributor of foods or beverages in the District of Columbia for immediate consumption on or about the place of business of such vendor or distributor shall permit any cup, glass, spoon, or fork that has been used for or in connection with the consumption of any such food or beverage to be used again for the same purpose until after it has been thoroughly washed in clean water.

Any person violating any of the provisions of Sec. 6c, Sec. 6d, Sec. 6e, or Sec. 6f, shall be punished by a fine of not less than one dollar nor more than twenty-five dollars for every such violation.—*Commissioners' Regulation of April 24, 1906.*

Sec. 7. That no person shall offer for sale within the District of Columbia any unwholesome, watered, or adulterated milk, or swill milk, or milk from cows kept up and fed on garbage, swill, or other deleterious substance; nor shall any person offer for sale within said District any butter or cheese made from such unwholesome milk; and any person violating the provisions of this section shall, upon conviction, be punished by a fine of not less than five nor more than twenty dollars for each and every such offense.

Sec. 12. Every manager of a store, market, cafe, lunch room, or of any other place where a food or a beverage is manufactured or prepared for sale, stored for sale, offered for sale, or sold, which store, cafe, lunch room, or other place is in operation at the time of the promulgation of this regulation, shall, on or before July 1, 1907, register his full name, and the location of said store, market, cafe, lunch room, or other place, and the nature of the business transacted, in a book to be kept in the health office for that purpose; and every manager of a store, market, cafe, lunch room, or other place where a food or beverage is manufactured or prepared for sale, stored for sale, offered for sale, or sold, that is first opened for business after the promulgation of this regulation shall, within five days after the opening of said store, market, cafe, lunch room, or other place, register in like manner. In event of a change in the manager or in the location of any store, market, cafe, lunch room, or other place aforesaid, the manager thereof shall call at the health office within five days after such change takes place and make a corresponding entry. Any person who violates the provision of this regulation shall, upon conviction thereof, be punished by a fine not exceeding twenty-five dollars for each and every such offense.—*Commissioners' Regulation of April 5, 1907.*

Sec. 13. Every manager of a store, market, dairy, cafe, lunch room, or any other place in the District of Columbia where a food, or a beverage, or confectionery, or any similar article, is manufactured or prepared for sale, stored for sale, offered for sale, or sold, shall cause it to be screened effectually, or effectually protected by power-driven fan or fans, so as to prevent flies and other insects from obtaining access to such food, beverage, confectionery, or other article, and shall keep such food, beverage, confectionery, or other article free from flies and other insects at all times. Any person violating the provisions of this regulation shall, upon conviction thereof, be punished by a fine of not more than twenty-five dollars for each and every such offense. This regulation shall take effect from and after the expiration of thirty days immediately following the date of its promulgation. —*Commissioners' Regulation of April 11, 1908.*

Sec. 14. Every manager of a store, market, dairy, cafe, lunch room, or of any other place in the District of Columbia where a food, or a beverage, or confectionery, or any similar article, is manufactured or prepared for sale, stored for sale, offered for sale, or sold, shall equip said store, market, dairy, cafe, lunch room, or other place, with running water, or other proper water supply if running water be not available, and with facilities and material for the proper washing, and shall cause such washing to be done, of the hands of all persons employed therein, and for the proper cleansing, and shall cause such cleansing to be done, of said store, market, dairy, cafe, lunch room, or other place, and of all apparatus, utensils, and materials used in connection therewith. Any persons violating the provisions of this regulation shall, upon conviction thereof, be punished by a fine of not more than twenty-five dollars for each and every such offense. This regulation shall take effect from and after the expiration of thirty days immediately following the date of its promulgation. *Commissioners' Regulation of May 31, 1907, as amended by orders of June 10, 1907, and April 11, 1908.*

Sec. 15. No person shall use any premises or any part of any premises in the District of Columbia for the preparation, manufacture, or storage for sale, or for the offering for sale, exchange, or delivery, of any food, drink, confectionery, or condiment for man, unless such premises or part of premises be provided with running water, or other proper water supply if running water be not available, and with all necessary facilities, apparatus, and material for the proper cleansing of said premises or part of premises, and of all apparatus, utensils, and materials used in connection therewith, and for the proper cleansing of the hands of all the persons employed therein. Any person violating any of the provisions of this section shall, upon conviction thereof, be punished by a fine of not more than twenty-five dollars for each and every such offense, and each day's violation shall be deemed to be a separate offense.—*Commissioners' Regulation of November 17, 1908.*

Regulations for the Prevention of the Spread of Scarlet Fever, Diphtheria, Measles, Whooping Cough, Chicken Pox, Epidemic Cerebro-Spinal Meningitis, and Typhoid Fever.

Sec. 5. No person residing in any dwelling house or in any apartments where there is in said dwelling house or apartments a patient suffering from diphtheria, scarlet fever, measles, or epidemic cerebro-spinal meningitis, shall, while so residing and during the continuance of such case, attend public or private school or Sunday school, or if the patient was suffering from diphtheria or scarlet fever, engage in the manufacture, preparation, storage, or sale of food or beverage.

Sec. 6. No person who has resided in any dwelling house or in any apartments while there was in such dwelling house or apartments a patient suffering from scarlet fever, diphtheria, measles, or epidemic cerebro-spinal meningitis shall after the removal, death, or recovery of the patient, or after the removal of such person from such dwelling house or apartments, attend public or private school, or Sunday school, or, if the patient was suffering from scarlet fever or diphtheria, engage in the manufacture, preparation, or storage of food or a beverage for sale, or in the sale of food or a beverage, without the written permission of the health officer, for a period following the first proper isolation of the patient, when no disinfection is to be made, and when disinfection is necessary immediately following the completion of such disinfection, as may be directed by the health officer, and continuing if the patient was suffering from scarlet fever, diphtheria, or epidemic cerebro-spinal meningitis, for seven days, or if the patient was suffering from measles, for fourteen days.—*Commissioners' Regulations of April 5, 1907.*

Establishing a Limit of Tolerance on Milk Bottles or Jars.

July 29, 1901.

Ordered: That the schedule of fees for inspecting and sealing glass bottles or jars used for the distribution or delivery of milk or cream to consumers, adopted June 17, 1901, and suspended July 1 and July 10, 1901, is hereby amended to read as follows, to take effect on and after the first of August, 1901:

That the glass bottles or jars used for the distribution or delivery of milk or cream to consumers, that hold, when filled to a level with the bottom of the cap or stopple, not less than seven ounces and six drams and not over eight ounces and two drams for one-half pint measure; not less than fifteen ounces and five drams and not over sixteen ounces and four drams for one pint; not less than thirty-one ounces and four drams and not over thirty-two ounces and four drams for one quart; not less than forty-seven ounces and three drams and not over forty-eight ounces and five drams for three pints; not less than sixty-three ounces and two drams and not over sixty-four ounces and six drams for one-half gallon, shall be sealed as measures and that all dealers in milk who use glass bottles or jars for the distribution or delivery of milk or cream to consumers shall be charged a fee of fifty cents per hundred bottles for such inspection and sealing.—(*Commissioners' order of July 29, 1901.*)

NOTICE TO LIBRARIANS AND BIBLIOGRAPHERS CONCERNING THE SERIAL PUBLICATIONS OF THIS LABORATORY.

The Hygienic Laboratory was established in New York, at the Marine Hospital on Staten Island, August, 1887. It was transferred to Washington, with quarters in the Butler Building, June 11, 1891, and a new laboratory building, located in Washington, was authorized by act of Congress, March 3, 1901.

The following *bulletins* [Bulls. Nos. 1–7, 1900 to 1902, Hyg. Lab., U. S. Mar. Hosp. Serv., Wash.] have been issued:

No. 1.—Preliminary note on the viability of the *Bacillus pestis*. By M. J. Rosenau.

No. 2.—Formalin disinfection of baggage without apparatus. By M. J. Rosenau.

No. 3.—Sulphur dioxid as a germicidal agent. By H. D. Geddings.

No. 4.—Viability of the *Bacillus pestis*. By M. J. Rosenau.

No. 5.—An investigation of a pathogenic microbe (*B. typhi murium* Danysz) applied to the destruction of rats. By M. J. Rosenau.

No. 6.—Disinfection against mosquitoes with formaldehyde and sulphur dioxid. By M. J. Rosenau.

No. 7.—Laboratory technique: Ring test for indol, by S. B. Grubbs and Edward Francis; Collodium sacs, by S. B. Grubbs and Edward Francis; Microphotography with simple apparatus, by H. B. Parker.

By act of Congress approved July 1, 1902, the name of the "United States Marine Hospital Service," was changed to the "Public Health and Marine-Hospital Service of the United States," and three new divisions were added to the Hygienic Laboratory.

Since the change of name of the Service the bulletins of the Hygienic Laboratory have been continued in the same numerical order, as follows:

No. 8.—Laboratory course in pathology and bacteriology. By M. J. Rosenau. (Revised edition March, 1904.)

No. 9.—Presence of tetanus in commercial gelatin. By John F. Anderson.

No. 10.—Report upon the prevalence and geographic distribution of hookworm disease (uncinariasis or anchylostomiasis) in the United States. By Ch. Wardell Stiles.

No. 11.—An experimental investigation of *Trypanosoma lewisi*. By Edward Francis.

No. 12.—The bacteriological impurities of vaccine virus; an experimental study. By M. J. Rosenau.

No. 13.—A statistical study of the intestinal parasites of 500 white male patients of the United States Government Hospital for the Insane; by Philip E. Garrison, Brayton H. Ransom, and Earle C. Stevenson. A parasitic roundworm (*Agamomermis culicis* n. g., n. sp.) in American mosquitoes (*Culex sollicitans*); by Ch. Wardell Stiles. The type species of the cestode genus *Hymenolepis*; by Ch. Wardell Stiles.

No. 14.—Spotted fever (tick fever) of the Rocky Mountains; a new disease. By John F. Anderson.

No 15.—Inefficiency of ferrous sulphate as an antiseptic and germicide. By Allan J. McLaughlin.

No. 16.—The antiseptic and germicidal properties of glycerin. By M. J. Rosenau.

No. 17.—Illustrated key to the trematode parasites of man. By Ch. Wardell Stiles.

No. 18. An account of the tapeworms of the genus *Hymenolepis* parasitic in man, including reports of several new cases of the dwarf tapeworm (*H. nana*) in the United States. By Brayton H. Ransom.

No. 19.—A method for inoculating animals with precise amounts. By M. J. Rosenau.

No. 20.—A zoological investigation into the cause, transmission, and source of Rocky Mountain "spotted fever." By Ch. Wardell Stiles.

No. 21. -The immunity unit for standardizing diphtheria antitoxin (based on Ehrlich's normal serum). Official standard prepared under the act approved July 1, 1902. By M. J. Rosenau.

No. 22.—Chloride of zinc as a deodorant antiseptic, and germicide. By T. B. McClintic.

No. 23.—Changes in the Pharmacopœia of the United States of America. Eighth Decennial Revision. By Reid Hunt and Murray Galt Motter.

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No. 25.—Illustrated key to the cestode parasites of man. By Ch. Wardell Stiles.

No. 26.—On the stability of the oxidases and their conduct toward various reagents. The conduct of phenolphthalein in the animal organism. A test for saccharin, and a simple method of distinguishing between cumarin and vanillin. The toxicity of ozone and other oxidizing agents to lapase. The influence of chemical constitution on the lipolytic hydrolysis of etheral salts. By J. H. Kastle.

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No. 30.—I. Maternal transmission of immunity to diphtheria toxin. II. Maternal transmission of immunity to diphtheria toxin and hypersusceptibility to horse serum in the same animal. By John F. Anderson.

No. 31.—Variations in the peroxidase activity of the blood in health and disease. By Joseph H. Kastle and Harold L. Amoss.

No. 32.—A stomach lesion in guinea pigs caused by diphtheria toxine and its bearing upon experimental gastric ulcer. By M. J. Rosenau and John F. Anderson.

No. 33.—Studies in experimental alcoholism. By Reid Hunt.

No. 34. I. *Agamofilaria georgiana* n. sp., an apparently new roundworm parasite from the ankle of a negress. II. The zoological characters of the roundworm genus *Filaria* Mueller, 1787. III. Three new American cases of infection of man with horsehair worms (species Paragordius varius), with summary of all cases reported to date. By Ch. Wardell Stiles.

No. 35. -Report on the origin and prevalence of typhoid fever in the District of Columbia. By M. J. Rosenau, L. L. Lumsden, and Joseph H. Kastle. (Including articles contributed by Ch. Wardell Stiles, Joseph Goldberger, and A. M. Stimson.)

No. 36.—Further studies upon hypersusceptibility and immunity. By M. J. Rosenau and John F. Anderson.

No. 37.—Index-catalogue of medical and veterinary zoology. Subjects: Trematoda and trematode diseases. By Ch. Wardell Stiles and Albert Hassall.

No. 38.—The influence of antitoxin upon post-diphtheritic paralysis. By M. J. Rosenau and John F. Anderson.

No. 39.—The antiseptic and germicidal properties of solutions of formaldehyde and their action upon toxines. By John F. Anderson.

No. 40.—Miscellaneous zoological papers. By Ch. Wardell Stiles and Joseph Goldberger.

No. 41.—Milk and its relation to the public health. By various authors.

No. 42. The thermal death points of pathogenic micro-organisms in milk. By M. J. Rosenau.

No. 43.—The standardization of tetanus antitoxin. An American unit established under authority of the act of July 1, 1902. By M. J. Rosenau and John F. Anderson.

No. 44.—Report No. 2 on the origin and prevalence of typhoid fever in the District of Columbia, 1907. By M. J. Rosenau, L. L. Lumsden, and Joseph H. Kastle.

No. 45.—Further studies upon anaphylaxis. By M. J. Rosenau and John F. Anderson.

No. 46.—*Hepatozoon perniciosum* (n. g., n. sp.); a hæmogregarine pathogenic for white rats; with a description of the sexual cycle in the intermediate host, a mite (*Lelaps echidinus*). By W. W. Miller.

No. 47.—Studies on thyroid: I. The relation of iodine to the physiological activity of thyroid preparations. By Reid Hunt and Atherton Seidell.

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No. 49.—Digest of comments on the United States pharmacopœia. Eighth decennial revision for the period ending December 31, 1905. By Murray Galt Motter and Martin I. Wilbert.

No. 50.—Further studies upon the phenomenon of anaphylaxis. By M. J. Rosenau and John F. Anderson.

No. 51.—Chemical tests for blood. By Joseph H. Kastle.

No. 52.—Report No. 3 on the origin and prevalence of typhoid fever in the District of Columbia. By M. J. Rosenau, L. L. Lumsden, and Joseph H. Kastle.

No. 53.—The influence of certain drugs upon the toxicity of acetanilide and antipyrine. By Worth Hale.

No. 54.—The fixing power of alkaloids on volatile acids and its application to the estimation of alkaloids with the aid of phenolphthalein or by the Volhard method. By Elias Elvove.

No. 55.—Quantitative pharmacological studies: adrenalin and adrenalin-like bodies. By W. H. Schultz.

No. 56.—Milk and its relation to the public health. (Revised and enlarged edition of Bulletin No. 41.) By various authors.

In citing these bulletins, beginning with No. 8, bibliographers and authors are requested to adopt the following abbreviations: Bull. No. ——, Hyg. Lab. U. S. Pub. Health & Mar. Hosp. Serv., Wash., pp. ——.

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The Service will enter into exchange of publications with medical and scientific organizations, societies, laboratories, journals, and authors. Its publications will also be sent to nonpublishing societies and individuals in case sufficient reason can be shown why such societies or individuals should receive them. All applications for these publications should be addressed to the "Surgeon-General, U. S. Public Health and Marine-Hospital Service, Washington, D. C."

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TREASURY DEPARTMENT
Public Health and Marine-Hospital Service of the United States

HYGIENIC LABORATORY.—BULLETIN No. 57

September, 1909

I. THE PRESENCE OF TUBERCLE BACILLI IN THE CIRCULATING BLOOD IN CLINICAL AND EXPERIMENTAL TUBERCULOSIS

By

JOHN F. ANDERSON

II. THE VIABILITY OF THE TUBERCLE BACILLUS

By

M. J. ROSENAU

WASHINGTON
GOVERNMENT PRINTING OFFICE
1909

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United States Public Health and Marine-Hospital Service.

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I. THE PRESENCE OF TUBERCLE BACILLI IN THE CIRCULATING BLOOD IN CLINICAL AND EXPERIMENTAL TUBERCULOSIS.[a]

By John F. Anderson,

Passed Assistant Surgeon and Assistant Director Hygienic Laboratory, United States Public Health and Marine-Hospital Service, Washington. D. C.

INTRODUCTION.

Clinicians and laboratory workers have long sought for aids in the diagnosis of incipient cases of pulmonary tuberculosis and obscure infections with the tubercle bacillus in other parts of the body. It is frequently impossible in the early stages of phthisis to find the bacilli in the sputum and often the physical signs are not sufficiently definite to warrant a positive diagnosis, and an early diagnosis means a probable good prognosis.

The various tuberculin reactions in adults are not always conclusive. So that any method that would enable us to make an early and positive diagnosis in a large percentage of cases would be of almost untold value to the clinician, health officer, and patient.

A recent paper by Rosenberger,[b] in which he reports the finding of tubercle bacilli in the blood of all cases examined, has excited much interest. If Rosenberger's claims are substantiated, they will be of great value in the diagnosis of tuberculosis. His statements were based largely upon finding in the blood bacilli possessing the morphological and tinctorial properties of the tubercle bacillus.

The method employed by him was to draw about 5 c. c. of blood from a vein in the arm and at once to mix it with an equal quantity of 2 per cent sodium citrate solution in normal salt solution. The mixture was well shaken and placed in the refrigerator for twenty-four hours, when smears were made from the sediment. The smears were then dried with moderate heat, placed in distilled water until complete laking of the blood, dried, fixed in a Bunsen flame and stained by the usual technic for tubercle bacilli. In a later paper he states that he used Pappenheim's solution as a decolorizer and counter stain.

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[a] Manuscript submitted for publication July 8, 1909.

[b] Rosenberger, Randle C. The presence of tubercle bacilli in the circulating blood in tuberculosis. Am. Journ. Med. Sci., vol. 137, No. 2, 1909, p. 267.

In his first paper Rosenberger reports positive results when 2 or 2.5 c. c. of the sediment were inoculated in two instances into guinea pigs.

Various observers since the time of Villemin have reported the finding of tubercle bacilli in the blood in tuberculosis.

Villemin[a] in 1868 reported the successful inoculation of a rabbit with tuberculosis by the blood collected from the femoral vein of a tuberculous rabbit; also, of a rabbit with the blood from a case of human tuberculosis.

I shall not attempt to give a review of the subject of tubercle bacilli in the blood, as the literature upon this subject up to 1904 has been well discussed by Bergeron[b] in his admirable thesis on the subject. In his paper Bergeron gives a fine critical review of previous work and in addition experiments by himself. In his conclusion he sums up the status of the subject so well that I shall quote the following from him:

1. The pathological anatomy shows that at a given moment the tubercle bacillus is able to pass into the general circulation, but the lesions capable of being interpreted as being the result of an infection in the blood are only to be encountered in acute tuberculosis.

2. Guinea pigs experimentally infected never present bacillemia either at the beginning of the infection or during the period of cachexia.

3. Rabbits experimentally infected usually present a kind of lesion which testifies to the passage of the bacilli into the blood at a given moment; but these bacillary discharges should be considered essentially transitory and ephemeral. In short, in spite of experiments to the contrary, it seems impossible to demonstrate the presence of the bacilli in the blood by means of the inoculation of guinea pigs as well in the beginning of the disease as later, when the infection has been made by subcutaneous or intraperitoneal injections.

In cases where the infection has been made intravenously the bacilli are localized more or less rapidly in the tissues and disappear from the circulation in a time which may not exceed four hours (Nocard), but may be as late as six days (Gaertner) or even later, though not much later.

4. The presence of tubercle bacilli in the blood of persons with miliary tuberculosis is a fact proved long ago, both by the pathological anatomy and autopsy and by examination of the blood during life. Researches, however, tend to show that the bacillemia of acute tuberculosis is a relatively rare phenomenon.

---

[a] Villemin: Etudes sur la tuberculose. Paris, 1868.

[b] Bergeron André: Etude critique sur la présence du bacille de Koch dans le sang. Thèse. Paris, 1904.

5. The inoculation of guinea pigs shews that in the course of non-miliary tuberculosis, even when it has assumed a rapid form, febrile or hemorrhagic, the bacillemia does not exist. The only case which we know of (Ettlinger) perhaps may be explained by the existence of a short premiliary stage terminating abruptly in this observation by a hyperacute peritonitis.

6. The results by the use of the inoscopic method of Jousset and the method of homogenization are not to be admitted without great reserve. These methods, in order to preserve their value, ought to be followed with a perfect asepsis very difficult to realize.

7. The most grave causes of error which are attached to the methods recently proposed for the direct finding of tubercle bacilli in the blood reside in the fact of the existence in our surroundings:

a. Of acid-resistant bacilli, well studied by Koch, Petri, Möeller, etc.

b. Of common saprophytic bacteria, which have acquired in the organic media where they are accidentally found, an acid-resistant power more or less accentuated.

TECHNIQUE EMPLOYED IN THE COLLECTION AND EXAMINATION OF THE BLOOD.

In order to endeavor to find out why the results of the older observers differed from those of Rosenberger, I undertook a study of tubercle bacilli in the blood.

My studies have been made on 48 cases of human tuberculosis and 13 guinea pigs and 8 rabblits experimentally infected. The study consisted of the examination of smears, cultures, and the inoculation of blood into fresh guinea pigs. With a few exceptions, which are noted in the protocols, the following procedure was employed:

In the human cases the blood was drawn from the vein at the bend of the elbow. Usually 10 or 20 c. c. of blood were taken and mixed with sufficient sodium citrate solution to give approximately a 1 per cent solution of the citrate, and thoroughly shaken. As soon as the blood was received at the laboratory it was divided into two lots. One lot was at once centrifuged for one hour at high speed. Two smears were made from the sediment, using in every case drawn-out pipettes that had never been used. Five drops of the sediment were then planted in a fermentation tube of lactose-bouillon and 5 drops on glycerin-potato; 2.5 c. c. of the sediment was next injected into the peritoneal cavity of a young guinea pig.

The cultures were placed in the incubator at 37° C.; the fermentation tube was examined for growth in twenty-four, forty-eight, and seventy-two hours, and discarded after that time. The potato cultures were sealed with paraffin and kept under observation for two months. The guinea pigs were weighed at intervals of a week.

Those that died were carefully autopsied, and if any lesions suggestive of tuberculosis was found an emulsion of the suspicious material was injected into another guinea pig. All animals alive at the end of two months were killed and autopsied.

The other half of the citrated blood was placed in the ice chest and at the end of twenty-four hours exactly the same procedure was followed with it as detailed above for the centrifuged portion.

The blood from the experimentally infected guinea pigs and rabbits was taken directly from the exposed heart after the animal had been etherized and, except in a few instances, treated as that from the human cases, though as a rule fermentation tube cultures were not made.

All the smears were first dried by moderate heat, then the blood was laked in distilled water, air dried, fixed in the Bunsen flame, stained with hot carbol-fuchsin, decolorized in 20 per cent sulphuric acid, washed in alcohol, counterstained with Loeffler blue, and examined for acid-fast bacilli.

I wish to acknowledge my indebtedness to Dr. W. H. Frost, who kindly collected most of the specimens of blood from the cases of human tuberculosis, and to Doctor Smith, resident physician of the district tuberculosis hospital, and his assistants, for courtesies extended Doctor Frost in the collection of the blood from the cases of human tuberculosis.

The results will be seen from the following protocols:

Blood from cases of human tuberculosis.

Case No.	History of case from which blood was taken.	Amount blood taken in citrate soda solution, c. c.	How blood was treated.	Acid-fast bacilli in sediment smears.	Growth tubercle bacilli on glycerin potato in 6 weeks.	Growth in fermentation tube in 72 hours.	Amount sediment intraperitoneally in guinea pig.	Guinea pig serial number.	Result. Death, in days later.	Result. Killed, days later.	Result. Autopsy findings.
							c. c.				
1 R. B.	Pulmonary tuberculosis, 2 [illegible] left apex [illegible] bacilli in sputum.	20	15 c. c. centrifuged.			...	5	1		70	No evidence of tuberculosis.
			5 c. c. in ice chest 24 hours.	[illegible]		....	2.5	2	27	...	Do.
2 G. S.	Pulmonary tuberculosis, 2 years' duration; infiltration [illegible] temperature nearly normal; improving; bacilli in sputum.	20	15 c. c. centrifuged.		—	[b] +	5	3	...	70	Do.
			5 c. c. in ice chest 24 hours.		—	—	2.5	4	..	72	Do.
[illegible] L. T.	[illegible] tum.	20	15 c. c. centrifuged.		–	—	5	5	.	70	Do.
			5 c. c. in ice chest 24 hours.		–	—	2.5	6	.	72	Do.
[illegible] A. W.	Pulmonary tuberculosis; 6 [illegible] tration l apex; stationary; bacilli in sputum.	12	7 c. c. centrifuged.		–	[c] +	3	6x		69	Do.
			5 c. c. in ice chest				2.5	7		72	Do.

[a] [illegible] acid-fast bacilli in [illegible]

[b] Hay bacillus.

[c] Coccus.

Blood from cases of human tuberculosis—Continued.

Case No.	History of case from which blood was taken.	Amount blood taken in citrate soda solution, c. c.	How blood was treated.	Acid-fast bacilli in sediment smears.	Growth tubercle bacilli on glycerin potato in 6 weeks.	Growth in fermentation tube in 72 hours.	Amount sediment intraperitoneally in guinea pig.	Guinea pig serial number.	Result. Death, in days, later.	Result. Killed, days later.	Result. Autopsy findings.
		c. c.					c. c.				
5 E. N.	Pulmonary tuberculosis; 5 years' duration; infiltration 1 apex; temperature usually normal; bacilli in sputum.	10	7 c. c. centrifuged.	–		–	3	8		72	No evidence of tuberculosis.
6 S. G.	Pulmonary tuberculosis; 6 months' duration; infiltration 1 apex; mild case; temperature normal; improving; bacilli in sputum.	20	15 c. c. centrifuged.			–	2.5	9	8	.	Do.
			5 c. c. in ice chest.				2.5	10	....	68	Do.
7 C. L.	Pulmonary tuberculosis; 5 months' duration, consolidation half 1 lung, fever 102° F.; bacilli in sputum.	20	15 c. c. centrifuged.		–		2.5	11		68	Do.
			5 c. c. in ice chest.				2.5	12		68	Do.
8 L. T.	Pulmonary tuberculosis; 1 year duration; extensive involvement 1 lung; fever 101° F.; bacilli in sputum.	20	15 c. c. centrifuged.		–		2.5	13		68	Do.
			5 c. c. in ice chest.				2.5	14		68	Do.
9 J. K.	Pulmonary tuberculosis; 6 months' duration; complete consolidation 1 lung, other infiltrated; temperature 102° F.; stationary; bacilli in sputum.	8	8 c. c. centrifuged.				2.5	15		68	Do.
10 A. G.	Pulmonary tuberculosis; 7 months' duration; consolidation 1 entire lung, other infiltrated; high temperature; bacilli in sputum.	7	7 c. c. centrifuged.		–	–	2.5	16		68	Do.
11 H. F.	Pulmonary tuberculosis; 5 months' duration; infiltration most of left lung; occasional elevation of temperature; bacilli in sputum.	20	15 c. c. centrifuged.				2.5	17	[illegible]		Do.
			5 c. c. in ice chest.				2.5	18	....	[illegible]	Do.
12 A. S.	Pulmonary tuberculosis; [illegible] months' duration; [illegible] extensive; [illegible] temperature; bacilli in sputum.	20	15 c. c. centrifuged.				2.5	[illegible]		[illegible]	Do.
			5 c. c. in ice chest.				2.5	20			Do.
13 J. W. C.	Pulmonary tuberculosis; 15 years' duration; [illegible] half 1 lung consolidated; temperature normal; bacilli in sputum.	[illegible]	15 c. c. centrifuged.				2.5	21		70	Do.
			5 c. c. in ice chest.				2.5	22			[illegible]
14 [illegible]	Pulmonary tuberculosis; [illegible] years' duration; [illegible] infiltration both [illegible] improving; [illegible] sputum.	[illegible]	5 c. c. centrifuged.				[illegible]	[illegible]		[illegible]	[illegible]
			5 c. c. in ice chest.				[illegible]	[illegible]		[illegible]	[illegible]
15 R. J.	Pulmonary tuberculosis; 1 year duration; [illegible] tion half of 1 lung; temperature [illegible] improving; bacilli in sputum.	10	[illegible] centrifuged.				[illegible]	[illegible]		[illegible]	Do.
			[illegible] ice chest.			–	2.5	26	....	[illegible]	[illegible]

Blood from cases of human tuberculosis—Continued.

Case No.	History of case from which blood was taken.	Amount blood taken in citrate soda solution (c. c.).	How blood was treated.	Acid-fast bacilli in sediment smears.	Growth tubercle bacilli on glycerin potato 6 weeks.	Growth in fermentation tube in 72 hours.	Amount sediment intraperitoneally in guinea pig.	Guinea pig serial number.	Result. Death, in days later.	Result. Killed, days later.	Result. Autopsy findings.
		c. c.					c. c.				
16 I. H. J.	Pulmonary tuberculosis; 8 months' duration; slight involvement; no consolidation; temperature normal; bacilli in sputum.	7	3.5 c. c. centrifuged.				3.5	27		69	No evidence of tuberculosis.
			3.5 c. c. in ice chest.	—			1.7	28		69	Do.
17 W. H.	Pulmonary tuberculosis; 2 years' duration; consolidation apex 1 lung; temperature nearly normal; bacilli in sputum.	5	2.5 c. c. centrifuged.	—			2.5	29		69	Do.
			2.5 c. c. in ice chest.				1.25	30	51	. .	Do.
18 J. B.	Pulmonary tuberculosis; 2 years' duration; slight involvement; improving; bacilli in sputum.	5.5	2.5 c. c. centrifuged.	-			2	31	.	69	Do.
			3 c. c. in ice chest.				1.25	32		69	Do.
19 J. H.	Pulmonary tuberculosis; 1 year duration; cavity in left lung; infiltration right lung; temperature 99° to 102° F.; bacilli in sputum.	6	5 c. c. centrifuged.				2.5	33		69	Do.
20 G. L. N.	Pulmonary tuberculosis; 2 years' duration; moderate involvement; discharged as improved; bacilli in sputum.	7	5 c. c. centrifuged.	-			2.5	34	....	69	Do.
21 C. S. F.	Pulmonary tuberculosis; 2 years' duration; 1 lung consolidated; temperature 100° F.; improving; bacilli in sputum.	10	5 c. c. centrifuged.			—	2.5	35	....	69	Do.
			5 c. c. in ice chest.			—	2.5	36	...	70	Do.
22 B. M.	Pulmonary tuberculosis; 6 months' duration; infiltration right apex; temperature 98° to 100° F.; improving; bacilli in sputum.	10	5 c. c. centrifuged.			—	2.5	37	3	...	Do.
			5 c. c. in ice chest.			—	2.5	38	....	69	Do.
23 [illegible]	Pulmonary tuberculosis; [illegible] months' duration; infiltration [illegible]; no fever; improving; bacilli in sputum.	10	5 c. c. centrifuged.		—	-	2.5	39	...	69	Do.
			5 c. c. in ice chest.			—	2.5	40	...	69	Do.
24 S. C.	Pulmonary tuberculosis; 3 months' duration; pneumopyothorax; temperature normal to 101° F.; improving; bacilli in sputum.	5	5 c. c. centrifuged.		-	—	2.5	41	...	69	Do.
25 [illegible]	Pulmonary tuberculosis; 1 year duration; infiltration [illegible]; no fever; [illegible] hospital; bacilli in sputum.	10	5 c. c. centrifuged.				2.5	42	....	69	Do.
			5 c. c. in ice chest.				2.5	43	....	69	Do.
26 E. B.	Pulmonary tuberculosis; 1 year duration; infiltration [illegible]; bacilli in sputum.	10	5 c. c. centrifuged.				2.5	44	.	69	Do.
			5 c. c. in ice chest.				2.5	45	.	69	Do.
27 [illegible]	Pulmonary tuberculosis; 9 months' duration; consolidation half left lung; fever [illegible]; improving; bacilli in sputum.	10	5 c. c. centrifuged.				2.5	46		69	Do.
			5 c. c. in ice chest.				2.5	47		68	Do.

Blood from cases of human tuberculosis—Continued.

Case No.	History of case from which blood was taken.	Amount blood taken in citrate soda solution c.c.	How blood was treated.	Acid-fast bacilli in sediment smears.	Growth tubercle bacilli on glycerin potato. Weeks.	Growth in fermentation tube in 72 hours.	Amount sediment intraperitoneally in guinea pig c.c.	Guinea pig serial number.	Result. Death, in days later.	Result. Killed, days later.	Result. Autopsy findings.
28 T. L.	Pulmonary tuberculosis, 2 months' duration; slight infiltration left lung; discharged as improved; bacilli in sputum.	10	5 c. c. centrifuged	-			2.5	48		68	No evidence of tuberculosis.
			5 c. c. in ice chest.				2.5	49		68	Do.
29 S. P.	Pulmonary tuberculosis; 3 years' duration; half 1 lung consolidated; temperature normal; improving; bacilli in sputum.	7	3.5 c. c. centrifuged.				2.5	50		68	Do.
			3.5 c. c. in ice chest.				2.5	51		67	Do.
30 P. D.	Pulmonary tuberculosis; 2 years' duration; 1 lung infiltrated; temperature 101° F.; bacilli in sputum.	10	5 c. c. centrifuged				2.5	52		68	Do.
			5 c. c. in ice chest				2.5	53		67	Do.
31 B. K.	Pulmonary tuberculosis; 3 years' duration; consolidation and cavity upper third 1 lung; arrested; no fever; bacilli in sputum.	10	5 c. c. centrifuged.				2.5	54		67	Do.
			5 c. c. in ice chest.				2.5	55		67	Do.
32 E. K.	Pulmonary tuberculosis; 3 years' duration; infiltration both apices; temperature 98° to 100° F.; bacilli in sputum.	8	4 c. c. centrifuged				2.5	56		68	Do.
			4 c. c. in ice chest				2.5	[illegible]		67	Do.
33 H. F.	Pulmonary tuberculosis; 8 years' duration; extensive infiltration; quiescent; fever 100° F.; bacilli in sputum.	5	5 c. c. centrifuged.				2.5	58	11		Do.
34 T. J. M.	Pulmonary tuberculosis; advanced; bacilli in sputum.	10	5 c. c. centrifuged.				2.5	59		67	Do.
			5 c. c. in ice chest.				2.5	60		67	Do.
35 C. W. S.	Pulmonary tuberculosis; 4 years' duration; extensive; tubercle bacilli in sputum; died 12 days later.	7	3.5 c. c. centrifuged				2.5	61		67	Do.
			3.5 c. c. in ice chest.	-			[illegible]	[illegible]		67	Do.
36 J. C.	Pulmonary tuberculosis; [illegible] months' duration; [illegible] consolidated, other infiltrated; not improving; bacilli in sputum.	10	[illegible] centrifuged.				[illegible]	[illegible]		67	Do.
			[illegible]				[illegible]	64		67	Do.
37 W. H.	Pulmonary tuberculosis; [illegible] year's duration; extensive involvement; cavity [illegible]; bacilli in sputum; died 16 days later.	[illegible]	[illegible] centrifuged				[illegible]	[illegible]		[illegible]	Do.
38 F. S.	Pulmonary tuberculosis; [illegible] years' duration; extensive involvement; bacilli in sputum; died [illegible] days later.	10	[illegible] centrifuged				2.5	[illegible]		[illegible]	Do.
			[illegible] ice chest.				[illegible]	[illegible]	28		Do.
			5 c. c. in [illegible] tubercle bacilli.				[illegible]	[illegible]	10		[illegible]

a The results from the blood [illegible] upon a possible bacteremia [illegible] As will be seen [illegible] potato or the infection of the [illegible] tuberculosis.

Blood from cases of human tuberculosis—Continued.

Case No.	History of case from which blood was taken.	Amount blood taken in c. c. into [illegible] solution c. c.	How blood was treated.	Acid-fast bacilli in sediment smears.	Growth tubercle bacilli on glycerin potato in 6 weeks.	Growth in fermentation tube in 72 hours.	Amount sediment intraperitoneally in guinea pig.	Guinea pig, serial number.	Result.		
									Death in days later.	Killed, days later.	Autopsy findings.
		c. c.					c. c.				
39 McC.	Tuberculosis of knee and elbow joints; tubercle bacilli not demonstrated.	20	5 c. c. centrifuged.				2.5	69		67	No evidence of tuberculosis.
			5 c. c. in icechest.				2.5	70		67	Do.
40 P. B. J.	Pulmonary tuberculosis; slight; incipient; bacilli in sputum.	10	5 c. c. centrifuged.				2.5	71		64	Do.
			5 c. c. in icechest.				2.5	72		[illegible]	Do.
41 E. H.	Pulmonary tuberculosis; 3½ years' duration; extensive involvement; bacilli in sputum; died 10 days later.	10	5 c. c. centrifuged.	-			2.5	73	31		Do.
			5 c. c. in icechest.	-			2.5	74	..	65	Do.
42 J. D.	Pulmonary tuberculosis; 6 months' duration; cavities both lungs; temperature 100° to 103° F.; bacilli in sputum.	8	4 c. c. centrifuged.			-	2.5	75	..	65	Do.
			4 c. c. in icechest.				2.5	76		65	Do.
43 G. V.	Pulmonary tuberculosis; 6 months' duration; moderately advanced; both lungs consolidated; temperature 98° to 101° F.; bacilli in sputum.	5	5 c. c. centrifuged.				2.5	77	...	64	Do.
44 [illegible]	Pulmonary tuberculosis; [illegible] years' duration; extensive; considerable temperature; bacilli in sputum.	[illegible]	5 c. c. centrifuged.				2.5	78	..	64	Do.
45 S. C.	Pulmonary tuberculosis; 5 months' duration; infiltration half 1 lung; bacilli in sputum; died [illegible] days later.	5	[illegible] c. c. centrifuged.			—	2.5	79	....	65	Do.
46 J. McC.	Pulmonary tuberculosis; 3 years' duration; slight involvement; no consolidation; temperature normal; bacilli in sputum.	20	15 c. c. centrifuged.				2.5	80	..	64	Do.
47 W. [illegible]	Pulmonary tuberculosis; 5 months' duration; general infiltration; fever 100° F.; bacilli in sputum.	25	10 c. c. centrifuged.				2.5	81		64	Do.
			5 c. c. in icechest.				2.5	82		64	Do.
48 [illegible]	Pulmonary tuberculosis; 6 months' duration; very extensive involvement; bacilli in sputum; died 2 [illegible]	6	[illegible] c. c. centrifuged.				2.5	83		64	Do.

BLOOD FROM RABBITS EXPERIMENTALLY INFECTED.

All the following rabbits from which blood was taken were inoculated subcutaneously with 0.5 c. c. of an emulsion of a glycerin potato growth of bovine tubercle bacilli. The rabbits were etherized at varying intervals after infection, the heart exposed and the blood drawn directly from the heart into a sterile syringe. In most instances a guinea pig was given 5 c. c. of the rabbit's heart blood intraperitoneally.

About 10 c. c. of the blood was added to sterile citrate of soda solution and placed in the ice chest overnight. Cultures were made on glycerin potato both from the whole heart's blood and from the sediment of the citrated blood. With a few exceptions two smears were made from the sediment of the citrated blood alone. After the blood was drawn from the heart the rabbit was killed and a careful autopsy made to determine if the animal was tuberculous. The details will be seen from the following protocols:

Rabbit No. 1. Blood drawn fourteen days after infection. No naked-eye evidence of tuberculosis at autopsy. No cultures made from the blood.

Five c. c. whole heart's blood injected into guinea pig No. 1A, weight 310 grams; killed seventy days later, weight 415 grams; generalized tuberculosis.

Rabbit No. 2. Blood drawn twenty-one days after infection. No naked-eye evidence of tuberculosis at autopsy.

Five c. c. whole heart's blood injected into guinea pig No. 2A, weight 290 grams; died two days later; no evidence of tuberculosis at autopsy. Glycerin potato culture of whole blood negative.

Ten c. c. blood in citrate soda solution in ice chest twenty-four hours. No tubercle bacilli found in sediment smears; no growth on glycerin potato in six weeks; 2.5 c. c. sediment intraperitoneally in guinea pig No. 2B, weight 285 grams; killed 61 days later, weight 350 grams; no evidence of tuberculosis at autopsy.

Rabbit No. 3. Blood drawn twenty-seven days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave growth of tubercle bacilli. Six c. c. whole heart's blood injected into guinea pig No. 3A, weight 300 grams; killed fifty-eight days later, weight 190 grams; general tuberculosis at autopsy.

Fourteen c. c. blood in citrate soda solution in ice chest twenty-four hours. No tubercle bacilli in sediment smears. Growth of tubercle bacilli from sediment on glycerin potato. Two and five-tenths c. c. sediment intraperitoneally into guinea pig No. 3B, weight 310 grams; died fifty-seven days later, weight 365 grams; general tuberculosis at autopsy.

Rabbit No. 4. Blood drawn twenty-seven days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Four c. c. whole heart's blood into guinea pig No. 4A, weight 310 grams; killed fifty-nine days later, weight 480 grams; general tuberculosis at autopsy.

Ten c. c. blood in citrate soda solution in ice chest twenty-four hours. No tubercle bacilli in sediment smears. Two and five-tenths c. c. sediment intraperitoneally into guinea pig No. 4B, weight 300 grams; killed fifty-eight days later, weight 475 grams; no evidence of tuberculosis at autopsy. No growth tubercle bacilli on glycerin potato in six weeks.

Rabbit No. 5. Blood drawn twenty-seven days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave growth of tubercle bacilli. Five c. c. whole heart's blood into guinea pig No. 5A, weight 320 grams; killed fifty-nine days later, weight 530 grams; general tuberculosis at autopsy.

Ten c. c. blood in citrate soda solution in ice chest twenty-four hours. No tubercle bacilli in sediment smears. Three c. c. sediment into guinea pig No. 5B, weight 295 grams; killed fifty-eight days later, weight 290 grams; general tuberculosis at autopsy. Growth of tubercle bacilli on glycerin potato from sediment.

Rabbit No. 6.—Blood drawn twenty-seven days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Five c. c. whole heart's blood into guinea pig No. 6A, weight 305 grams; killed fifty-nine days later, weight 550 grams; no evidence of tuberculosis at autopsy.

Ten c. c. blood in citrate soda solution in ice chest twenty-four hours. No tubercle bacilli in sediment smears. Two and five-tenths c. c. sediment intraperitoneally in guinea pig No. 6B, weight 310 grams; killed fifty-eight days later, weight 480 grams; general tuberculosis. No growth of tubercle bacilli from sediment on glycerin potato.

Rabbit No. 7. Blood drawn twenty-seven days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave growth of tubercle bacilli. Four c. c. whole heart's blood into guinea pig No. 7A, weight 315 grams; died fifty-seven days later, weight 420 grams; general tuberculosis at autopsy.

Ten c. c. blood in citrate soda solution in ice chest twenty-four hours. No tubercle bacilli in sediment smears. Two and five-tenths c. c. sediment intraperitoneally into guinea pig No. 7B, weight 300 grams; died fifty-seven days later, weight 310 grams; general tuberlosis at autopsy. Growth of tubercle bacilli from sediment on ycerin potato.

Rabbit No. 8.—Blood drawn twenty-seven days after infection. eneral tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle cilli. Five c. c. whole heart's blood into guinea pig No. 8A, weight 0 grams; killed fifty-nine days later, weight 415 grams; general berculosis at autopsy.

Ten c. c. blood in citrate soda solution in ice chest twenty-four urs. No tubercle bacilli in sediment smears. Four c. c. sediment traperitoneally in guinea pig No. 8B, weight 300 grams; died fifty-ven days later, weight 420 grams; general tuberculosis at autopsy. growth of tubercle bacilli from sediment on glycerin potato.

BLOOD FROM GUINEA PIGS EXPERIMENTALLY INFECTED.

All the following guinea pigs from which blood was taken were oculated subcutaneously with five-tenths c. c. of an emulsion of glycerin potato growth of bovine tubercle bacilli. The guinea gs were etherized at varying intervals after infection, the heart posed and the blood drawn directly from the heart into a sterile ringe with citrate of soda solution. Usually about five c. c. of e whole citrated blood was at once injected into the peritoneal vity of a fresh guinea pig.

The remainder of the blood in the syringe was placed in the ice est over night.

Cultures were made on glycerin potato both from the whole heart's ood and from the sediment of the citrated blood. With a few ceptions two smears were made from the sediment of the citrated ood alone. After the amount of blood desired had been taken om the heart the guinea pig was killed and a careful autopsy made determine if the animal was infected with tuberculosis. The tails will be seen from the following protocols:

Guinea pig No. 100.—Blood drawn fourteen days after infection. neral tuberculosis at autopsy. No cultures made from the blood.

Five c. c. whole heart's blood injected into guinea pig No. 100A, ight 305 grams; killed sixty-seven days later, weight 450 grams; evidence of tuberculosis at autopsy.

Guinea pig No. 101.—Blood drawn fourteen days after infection. neral tuberculosis at autopsy. No cultures made from the blood.

Four c. c. whole heart's blood into guinea pig No. 101A, weight 0 grams; killed sixty-seven days later, weight 510 grams; no evi-nce of tuberculosis at autopsy.

Guinea pig No. 102.—Blood drawn sixteen days after infection. eneral tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Four c. c. whole heart's blood into guinea pig No. 102A, weight 290 grams; died ten days later; no evidence of tuberculosis at autopsy.

Five c. c. blood in citrate solution in ice chest over night. Five c. c. sediment into guinea pig No. 102B, weight 310 grams; killed sixty-nine days later, weight 530 grams; no evidence of tuberculosis at autopsy. No growth of tubercle bacilli from sediment on glycerin potato. No tubercle bacilli found in sediment smears.

Guinea pig No. 103.—Blood drawn sixteen days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Four c. c. whole heart's blood into guinea pig No. 103A, weight 300 grams; killed sixty-nine days later, weight 435 grams; no evidence of tuberculosis at autposy.

Five c. c. blood in citrate solution in ice chest over night. Five c. c. sediment into guinea pig No. 103B, weight 275 grams; killed sixty-eight days later, weight 525 grams; no evidence of tuberculosis. No growth of tubercle bacilli from sediment on glycerin potato. No tubercle bacilli in sediment smears.

Guinea pig No. 104.—Blood drawn eighteen days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Five c. c. whole heart's blood into guinea pig No. 104A, weight 285 grams; died eleven days later; no evidence of tuberculosis at autopsy.

Five c. c. blood in citrate solution in ice chest over night.

Two and five-tenths c. c. sediment into guinea pig No. 104B, weight 280 grams; died eleven days later; no evidence of tuberculosis at autopsy. No growth on glycerin potato. Tubercle bacilli not found in sediment smears.

Guinea pig No. 105.—Blood drawn eighteen days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Five c. c. whole heart's blood in guinea pig No. 105A, weight 280 grams; killed sixty-seven days later, weight 500 grams; tubercles in spleen, liver, lungs, and on peritoneum.

Five c. c. heart's blood in citrate solution in ice chest over night. No tubercle bacilli in sediment smears. No growth from sediment on glycerin potato.

Two and five-tenths c. c. sediment into guinea pig No. 105B, weight 280 grams; killed sixty-six days later, weight 530 grams; general tuberculosis at autopsy.

Guinea pig No. 106.—Blood drawn twenty-one days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Five c. c. whole heart's blood into guinea pig No. 106A, weight 280 grams; killed sixty-five days later, weight 560 grams; no evidence of tuberculosis at autopsy.

Five c. c. blood in citrate solution in ice chest over night. No tubercle bacilli in sediment smears. No growth from sediment on glycerin potato. Two and five-tenths c. c. sediment in guinea pig No. 106B, weight 240 grams; killed sixty-two days later, weight 335 grams; no evidence of tuberculosis at autopsy.

Guinea pig No. 107. Blood drawn twenty-one days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Five c. c. whole heart's blood in guinea pig No. 107A, weight 300 grams; killed sixty-three days later, weight 460 grams; no evidence of tuberculosis at autopsy.

Five c. c. blood in citrate solution in ice chest over night. No tubercle bacilli in sediment smears. No growth from sediment on glycerin potato. Two and five-tenths c. c. sediment in guinea pig No. 107B, weight 290 grams; killed sixty-three days later, weight 480 grams; no evidence of tuberculosis at autopsy.

Guinea pig No. 108. Blood drawn twenty-five days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato showed no growth of tubercle bacilli. Eight c. c. whole heart's blood in guinea pig No. 108A, weight 290 grams; killed sixty days later, weight 485 grams; no evidence of tuberculosis at autopsy.

Guinea pig No. 109. Blood drawn twenty-five days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato showed no growth of tubercle bacilli. Five c. c. whole heart's blood in guinea pig No. 109A, weight 280 grams; died seven days later; no evidence of tuberculosis at autopsy.

Guinea pig No. 110. Blood drawn twenty-five days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Five c. c. whole heart's blood into guinea pig No. 110A, weight 290 grams; killed sixty days later, weight 525 grams; no evidence of tuberculosis at autopsy.

Guinea pig No. 111. Blood drawn twenty-five days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Five c. c. whole heart's blood into guinea pig No. 111A, weight 285 grams; killed sixty days later, weight 510 grams; no evidence of tuberculosis at autopsy.

Guinea pig No. 112. —Blood drawn twenty-five days after infection. General tuberculosis at autopsy.

Whole heart's blood on glycerin potato gave no growth of tubercle bacilli. Eight c. c. whole heart's blood into guinea pig No. 112A, weight 280 grams; died sixty days later, weight 450 grams; general tuberculosis at autopsy.

SUMMARY AND DISCUSSION.

An examination of the above experiments shows that there were 47 cases of pulmonary tuberculosis in which tubercle bacilli were found in the sputum and one case of joint tuberculosis in which the bacilli were not found. From these 48 cases of human tuberculosis glycerin-potato cultures, guinea-pig inoculations, and smears were made from the sediment obtained by centrifugation; but in not a single instance were tubercle bacilli demonstrated either by the examination of the smears, cultures, or animal inoculations.

Glycerin-potato cultures, guinea-pig inoculations, and smears were made in 35 of the 48 cases from the sediment obtained by placing the citrated blood in the ice chest for twenty-four hours. In one instance (case No. 1) the smears showed an organism that had the morphological and tinctorial appearances of tubercle bacilli; but the glycerin-potato culture and guinea-pig inoculations were negative. The smears made from the centrifugalized sediment in the same case were negative.

Thirty-five of the 48 cases had smears, cultures, and animal inoculations made from the sediment obtained both by the centrifuge and by gravity for twenty-four hours in the ice chest.

In only a few instances was there a growth in the fermentation tubes in seventy-two hours, showing that by care in the collection and handling of the blood contamination may be avoided in the great majority of cases.

If tubercle bacilli had been present in sufficient numbers in the blood to be detected in smears, it would certainly seem that they should have been found in more than one instance in this series of 48 cases, in 47 of which tubercle bacilli had been found in the sputum. In most cases 2.5 c. c. of the sediment, either obtained by the centrifuge or by gravity, or both, were inoculated into guinea pigs, and this amount was many times the amount used to make the smears, yet all the animals inoculated were negative.

The glycerin-potato cultures were also uniformly negative. That the failure to find tubercle bacilli in the blood from these cases by animal inoculation, even when not found in smears, was not due to a bactericidal action of the serum is negatived by case No. 38 and by the experiments with the blood of rabbits experimentally infected. To a portion of the citrated blood from case No. 38 a very small amount of an emulsion of tubercle bacilli was added; tubercle bacilli were demonstrated by smears, culture, and guinea-pig inoculations, showing that the bactericidal action, if any, was very slight.

The smears in every instance were carefully examined by two and a portion of them by three different observers. I am satisfied that if acid-fast bacilli had been present they would not have escaped detection.

The blood from 8 rabbits experimentally infected was examined for tubercle bacilli. The blood from 7 of the 8 was examined by glycerin-potato cultures and guinea-pig inoculations of the whole heart's blood and of the sediment from the citrated blood in the ice chest for twenty-four hours. The blood from one rabbit (No. 1) was examined only by injection of 5 c.c. into a guinea pig.

Smears were made from the sediment of the citrated blood in the ice chest of 7 of the rabbits. As the blood of a number of the rabbits was proved by cultures and guinea-pig inoculations to contain tubercle bacilli, an unusually thorough search was made for acid-fast bacilli in the sediment smears of these cases. Two smears were made from the blood of each rabbit. The smears were examined by three trained laboratory men, but not in a single instance were tubercle bacilli found, though the blood contained tubercle bacilli in sufficient numbers to grow on potato and to infect guinea pigs.

Six of the 8 rabbits showed naked-eye appearances of tuberculosis at the autopsy made after the blood was taken. The blood of 1 of the 2 remaining was infective, even though there were no naked-eye lesions of tuberculosis.

Tubercle bacilli grew on the glycerin potato in 3 of the 6 cases, both from the whole heart's blood and from the citrated sediment. The blood was infective for guinea pigs by both methods in 1 of the 6, yet tubercle bacilli could not be found in the smears.

These results with the blood of rabbits are strongly against the idea that the blood serum of individuals with tuberculosis has a bacteriolytic power sufficient to lessen or destroy the virulence of tubercle bacilli in the blood stream so that, while they may be found in smears, they will not grow on cultures or infect animals.

The blood from only one guinea pig (No. 105) experimentally infected was found to contain tubercle bacilli by inoculation into fresh animals. The blood from all the others was negative, both culturally and by inoculation. No tubercle bacilli were found in the smears made from the citrated blood of No. 105 in the ice chest over night, though it was infective for the fresh animal.

It is of interest to note that the blood of only 1 guinea pig out of 13 contained tubercle bacilli, while that of 7 out of 8 rabbits experimentally infected contained them in sufficient numbers to be demonstrated either culturally or by inoculation.

There are several interesting points that come out of the above results. The first is that acid-fast bacilli were found only once in smears made from the blood of the 48 cases of human tuberculosis, although the bacilli had been demonstrated in the sputum of 47 of them. In the one case (case No. 1) acid-fast bacilli were not found in the centrifugalized sediment, but were found in the sediment of the citrated blood that had remained in the ice chest over night.

The question is, Were these acid-fast bacilli tubercle bacilli? If so, why did they not grow on cultures and infect guinea pigs? I must confess that I am unable to answer these questions, but am inclined to the opinion that they were not tubercle bacilli for the following reasons: The acid-fast bacilli were present in the smears in such numbers as to be seen in practically every field; they were most frequently seen in clumps consisting of 3 to 30 or more bacilli; most of the individual bacilli were shorter than the usual type of human tubercle bacilli. Now, the smear was made from one large drop of the citrated sediment and from the numbers seen in the portion of the slide examined there must have been many hundreds of bacilli in each drop of the sediment. Five drops of the same sediment were planted on glycerin potato, but no growth resulted, and 2.5 c. c. of the same sediment was inoculated into a guinea pig (guinea pig No. 2), but with negative results for tuberculosis. These amounts were many times that used to make the smear and, of course, must have contained a great many more bacilli; but tubercle bacilli were not demonstrated by cultures or animal inoculation.

As mentioned before, the acid-fast bacilli were not found in the smears made from the centrifugalized sediment. The case from which the blood was taken was in a very early stage and was improving at the time the blood was taken; had no fever, no night sweats, very slight cough, and was gaining in weight, but still had bacilli in the sputum at that time.

His occupation being that of a stableman, and being employed as such at the time the blood was taken, might suggest a possible contamination of the blood with an acid-fast grass or hay bacillus on the skin, which developed in the citrated blood in the ice chest over night and was found in the smears made from the sediment. This possibility emphasizes the great care necessary to avoid the entrance of acid-fast bacilli from any source, such as the skin, air, water, and citrate of soda, etc., into the blood when it is drawn, and is, perhaps, in harmony with the latter part of Bergeron's conclusions.

The second point is, that in my hands the finding of tubercle bacilli in smears made from the blood of known cases of tuberculosis on account of almost uniformly negative results seem to be of little value as an aid in the diagnosis of tuberculosis. These negative results could not have been due to the tubercle bacilli being overlooked, for all the smears were examined by at least two observers and a part of them by three, and, moreover, cultures and animal inoculations were negative.

The third point brought out by the experiments is that tubercle bacilli were demonstrated in the blood of 7 of the 8 rabbits experimentally infected. In 3 of the 7 they were found both by cultures and by animal inoculations. From this it would seem that when rabbits are infected by subcutaneous inoculation of tubercle bacilli the latter are found in the circulating blood in a large proportion of the cases, but not in sufficient numbers to be detected in smears. It is of especial interest to note that the blood of a rabbit (No. 1), which at the autopsy did not present any naked-eye evidences of tuberculosis, was infective for a guinea pig.

Tubercle bacilli were found in the blood of only 1 guinea pig experimentally infected. It seems, therefore, that whereas tubercle bacilli are frequently found in the blood of tubercular rabbits, it is unusual to find them in the blood of tubercular guinea pigs and humans in numbers or virulence sufficient to infect fresh animals.

The culture of tubercle bacilli from the blood of the experimentally infected rabbits is one of the few, if not the first, recorded instances in which tubercle bacilli have been grown from the blood of tubercular animals.

II. THE VIABILITY OF THE TUBERCLE BACILLUS.

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Practically all observers agree with Koch that human sputum is the main source of human tuberculosis. Whether the tubercle bacillus is usually transferred directly or indirectly, in moist or in dry state, by inhalation or ingestion, are questions still at issue.

The viability of the tubercle bacillus, therefore, assumes a special significance in aiding the solution of problems concerning the channels of infection and methods of transmission of this disease. Further, a correct understanding of the vitality of the tubercle bacillus is of first importance in carrying out many of our prophylactic measures.

No one questions that many of the tubercle bacilli are still alive and virulent in the droplets that are sprayed from the mouths of consumptive persons. Likewise many of the bacilli must be fresh and active when deposited by consumptives upon drinking cups, spoons, towels, or other articles mouthed by well persons shortly afterwards and without intervening cleansing. Whether the bacilli remain alive and virulent in dried and pulverized sputum in sufficient numbers to frequently form a dangerous part of the dust of rooms and streets is a question still open for solution.

Two principal destructive agencies act on the tubercle bacillus in sputum expectorated on streets and out-of-door places: (1) Desiccation and (2) the action of light. The bacillus also has to contend with other destructive agencies, such as the putrefactive and other organisms in the sputum or gaining access to it.

THE TUBERCLE BACILLUS AND ITS SUPPOSED SPORE.

Koch at first believed that the tubercle bacillus contained spores. He was led into this error by the beaded appearance of the rod. We now know that the tubercle bacillus does not possess an endogenous spore, such as that of the anthrax bacillus, the tetanus bacillus, or the hay bacillus.

Two circumstances led early investigators into wrong conclusions as to the viability of the tubercle bacillus. One was this belief that the organism contained spores. The other was ignorance of the role played by dead tubercle bacilli. Some of the earlier workers spent much time in trying to differentiate the resistance of the tubercle bacillus with and without spores. These earlier results are still quoted, without qualification, in many of the recent text-books and writings upon the subject. The notion that the tubercle bacillus has a spore is not altogether dead and has created an exaggerated idea of its hardiness and resistance.

THE TUBERCLE BACILLUS AND ITS WAXY SUBSTANCE.

It was soon found that although the tubercle bacillus does not have a true spore, it contains an unusually large quantity of waxy substance fats and fatty acid.[1] It is supposed that these fatty substances surround the rod, thus affording it a protecting covering. This envelope is supposed to hinder drying and also to protect the bacillus against the direct injurious influences of various external agencies.

The presence of the unusual amount of fatty and waxy substances led to a revision of our views upon the subject of the viability of the bacillus by placing it in an isolated and intermediate position between the spore-bearing and the nonspore-bearing rods. As to just how far this view is justified will be discussed subsequently.

ACID-FAST PROPERTY.

The difficulty with which the tubercle bacillus takes the basic aniline dyes and the great tenacity with which it holds them are noteworthy. True spores also stain with difficulty and resist the decolorizing action of acids. The analogy between the staining and decolorizing property of spores and the tubercle bacillus is not infrequently mentioned in the literature as indicating far greater resistance on the part of the tubercle bacillus to heat, dryness, putrefaction, chemicals, etc., than is the case with the great run of nonspore-bearing organisms. Except for the analogy, there is no indication that there is a definite relation between the viability of an organism and the ease or difficulty with which it stains and decolorizes. In fact, some protozoa, certain spirochetes, and other frail micro-organisms stain with more or less difficulty or require special technique.

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[1] Twenty-[illegible] and two-tenths per cent according to Hammerschlag; 22 per cent, Klebs; 37 per cent, de Schweinitz and Dorset; 20 to 25 per cent, Koch and Aronson; from 8 and 10 to 25 and 26 per cent, Ruppel, expressed in terms of alcohol-ether extract. This consists of fatty acids and neutral fat and a waxy substance. In contrast to the tubercle bacillus, other bacteria contain only 1.7 to 7 and 10.1 per cent alcohol-ether extractives. According to Aronson the tubercle wax is, for the most part, not in the bacillus, but between the bacilli as a product of secretion.

THE TRUE POSITION OF THE TUBERCLE BACILLUS.

Some of the more recent work upon the viability of the tubercle bacillus indicates that it has little if any greater resistance to heat, dryness, putrefaction, chemicals, sunlight, and other injurious agencies than the great bulk of nonspore-bearing bacteria. Its usual habitat in albuminous, mucoid, or fatty substances, such as pus, mucopus, necrotic material, milk, etc., protects it to a certain extent. This protection, however, is an accident of its environment rather than an inherent vital property.

We now know that 60° C. for twenty minutes is sufficient to kill the tubercle bacillus in milk, bouillon, water, and other fluids.[a] At one time it was believed that the tubercle bacillus resisted boiling temperatures. Experiments have resulted in gradually reducing the accepted thermal death point, until now we can state with considerable confidence that the tubercle bacillus has very little more resistance than a number of other nonspore-bearing bacteria; for instance, the typhoid bacillus, the dysentery bacillus, and the colon bacillus.

Some writers seem to consider it rather remarkable that the tubercle bacillus—an organism without a spore—should live several months in dust, in water, on fabrics, in sputum, etc. For instance, Schill and Fischer found it alive one hundred and eighty-six days in dried sputum; Toma virulent up to ten months; Sawizky dead after two and a half months; Twitchell found it virulent on a handkerchief seventy days but dead in one hundred and ten days; Rickards, Slack, and Arms found it virulent after eighty-five days, kept dark and dry; and Feltz found dried sputum in a room virulent up to seven and nine months.

It is possible, however, to cite from the literature instances of other nonspore-bearing organisms living similar periods of time under like conditions. For instance, observers have found that the cholera spirillum may live for one hundred and fifty to two hundred days when dried under favorable conditions. Sirena and Alessi report that such a frail organism as the *Diplococcus pneumoniæ* did not die until after one hundred and ninety-two days when dried on silk threads and kept in a moist room, and one hundred and sixty-four days in a dry room.

In some experiments on the plague bacillus I found it to live over four months on a piece of dry sponge and over three months in water under laboratory conditions.[b] Similar instances might be multiplied.

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[a] Rosenau, M. J. The thermal death points of pathogenic micro-organisms in milk. Hyg. Lab. Bull. No. 42, U. S. Public Health and Marine-Hospital Service, Washington, 1908, pp. 85. 8°.

[b] Rosenau, M. J. Viability of the *Bacillus pestis*. Hyg. Lab. Bull. No. 4, U. S. Marine-Hospital Service, Washington, 1901.

On the other hand, the following instances are given of experimenters who report a relatively short vitality for the tubercle bacillus dried in sputum not exposed to direct sunlight: Cadéac found it dead in ten days and sometimes less; Hill, sixteen days; Ransom and Delepine, nineteen days; and Galtier, after thirty days.

The question at issue from a practical standpoint is not how long a few tubercle bacilli may occasionally survive in dried sputum, but how soon they usually die or become harmless. In order to determine this point much more data will be required than are now available.

All authorities who have worked upon the subject seem agreed that the tubercle bacillus loses its virulence before it finally dies. This is also the case with other pathogenic nonspore-bearing bacteria.

It seems that the more carefully the tubercle bacillus is studied the nearer its viability is seen to approach that of other bacteria of its class.

CRITERION OF DEATH.

It is difficult to determine just when a microorganism dies. This difficulty is increased in the case of the tubercle bacillus. The reluctance with which this organism grows upon artificial media makes cultural methods totally inapplicable; it is necessary to resort to animal inoculations. Here again we meet an unusual difficulty, for the dead tubercle bacillus may produce lesions indistinguishable from those which are caused by live cultures.

A study of the literature shows that much of the work upon the viability of the tubercle bacillus has little value for lack of a clear criterion of death. Many experiments will be found recorded in which the tubercle bacillus is exposed to various influences, such as drying, heat, chemicals, sunlight, putrefaction, etc., and after a certain period of time the dried material is inoculated into rabbits or guinea pigs. In about two months after the inoculation the animal is usually killed, and if caseous abscesses and other lesions about the site of inoculation are found and the tubercle bacillus is rediscovered on stained smears made from the local lesions, the conclusion is drawn that the tubercle bacillus is alive, and the results are recorded as positive.

It is now well known that the dead tubercle bacilli may cause lesions characterized by giant cells, caseous necrosis, etc. I have found that moderately large amounts of tubercle cultures surely killed by heat (100 for one hour) may cause very extensive lesions when injected into the peritoneal cavities of guinea pigs. So closely do these lesions sometimes resemble those produced by the living bacteria that unless the portions of the spleen, liver, lungs, glands, and local processes are inoculated into another animal it is not possible to know whether we are dealing with the effects of living or of dead bacteria. When the lesions are plainly generalized throughout the substance of the liver, spleen, lungs, etc., these secondary inoculations may not be necessary.

The various organs (whether they contain macroscopic lesions or not), bits of tissue, and parts of lesions should be ground up with salt solution and the extract injected into the peritoneal cavity of a normal ("secondary") guinea pig; if the bacteria are alive there will develop a generalized fatal tuberculosis; if dead, only a trifling slight localized process. This method is time consuming and expensive, but is often indispensable as a final test.

It is well known that tuberculin can not distinguish the lesions produced by live tubercle bacilli from those produced by dead ones. In a series of experiments on this point I found that three out of eight guinea pigs with lesions produced by dead tubercle bacilli died as the result of a subcutaneous injection of 2 c. c. of tuberculin (O. T.).[a]

AN EXPLANATION OF DISCREPANCIES IN PUBLISHED REPORTS.

When one reads the literature upon the subject of the viability of the tubercle bacillus he is struck first by the extravagant claims made by some investigators for the prolonged life and unusual resistance of the tubercle bacillus. There are also marked discrepancies in the results obtained by different experimenters with apparently well-planned and carefully controlled work.

It seems that one of the main reasons for these variations is the failure to take note of the pathogenic power of the dead tubercle bacillus. In 1884 Schill and Fischer studied the action of a temperature of 100° C upon tuberculous sputum. They reported that 100° C. of dry heat did not surely destroy the virulence of tuberculous sputum, whether dry or moist. These experiments are frequently quoted to show that the tubercle bacillus resists boiling temperatures; but we now know that the thermal death point of the tubercle bacillus is considerably less than that indicated by earlier work.

Schill and Fischer also found that the bacillus retained life in sputum dried one hundred and forty-three days, but was not infective after one hundred and seventy-nine days. The method used consisted in inoculating guinea pigs, but the authors do not give the details of the experiments.

In view of the fact that earlier investigators fell into error concerning the thermal death point of the tubercle bacillus, it may be assumed that a repetition of their work upon the viability of the organism when dried in sputum, etc., may cause us to revise our views upon the subject.

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[a] Rosenau, M. J.: The thermal death points of pathogenic microorganisms in milk. Hyg. Lab. Bull. No. 42, U. S. Public Health and Marine-Hospital Service, Washington, 1908, pp. 85. 8°.

According to the work of Kitasato most of the tubercle bacilli in sputum, in cavities, and even in the tissues, are dead. This opinion was based largely upon negative results obtained by cultural methods. As our present cultural methods do not give trustworthy evidence upon the viability of the tubercle bacillus, further work upon the subject is necessary, especially as Straus confirmed the inadequacies of such methods, particularly in obtaining first cultures of tubercle bacilli. A certain acclimatization is necessary for their ready growth upon artificial media. It is true that Kitasato claims to have seen, in some cases, inoculation into guinea pigs fail, but Straus and Park have made analogous experiments with opposite results.

THE SHORT LIFE OF CULTURES CONTRASTED WITH THE LONG LIFE UNDER UNFAVORABLE CONDITIONS.

The comparatively short life of pure cultures of the tubercle bacillus under most favorable conditions contrasts strangely with the long life claimed for the organism under unfavorable conditions. It appears that the life of the tubercle bacillus in cultures is shorter the more rapid and more luxuriant the growth.

Cornet states that serum cultures remain alive about six months; glycerin-agar cultures are often partially or wholly dead in six to eight weeks. It appears that cultures of avian tubercle retain their vegetability and pathogenic power much longer; according to Maffucci, even two years. Straus states that cultures of human tubercle are only exceptionally capable of reproduction after five to six months; after eight to twelve months they fail regularly. The virulence of the cultures likewise diminishes with age. According to Theobald Smith, tubercle cultures are usually dead after three months.

THE THERMAL DEATH POINT.

In a recent publication[a] I reported the results of my experiments upon the thermal death points of pathogenic micro-organisms in milk. Nine experiments were made with five different cultures of the bovine tubercle bacillus. The conclusion was drawn that the tubercle bacillus in milk loses its infective properties for guinea pigs when heated to 60° C. and maintained at that temperature for twenty minutes, or to 65° C. for a much shorter time.

It should be remembered that the milk in these tests was very heavily infected with virulent cultures, as indicated by the prompt death of the control animals. Milk would practically never contain such an enormous amount of infection under natural conditions. It is justifiable to assume that if 60° C. for twenty minutes is sufficient to destroy the infectiveness of such milk when injected into the peritoneal cavity of a guinea pig, any ordinary market milk after such treatment would be quite safe for human use by the mouth so far as tubercle bacilli are concerned.

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[a] Hyg. Lab. Bull. No. 42, U. S. Public Health and Marine-Hospital Service. A complete [illegible] of the literature upon the subject will be found upon pp. 34-47 of the above publication.

It is difficult, if not impossible, to summarize the work of others upon the thermal death point of the tubercle bacillus. The following table necessarily leaves out many factors:

Showing the thermal death point of the tubercle bacillus as found by various investigators.

Investigator	Killed at	Not killed at–
Martin, 1882		80°.
May, 1883	By cooking	
Sormani, 1884	Boiling 5 minutes	90° for 10 minutes.
Schill and Fisher, 1884		100°.
Voelsch, 1887		100°, boiling twice.
Yersin, 1888.	60°, 10 minutes (—spores) 70°, 10 minutes (+ spores)	
Bitter, 1890	68°, 20 minutes.	
Bang, 1891	70°, 5 minutes enfeebles 75°, 5 minutes (sometimes enfeebles) 80°, (sometimes kills) 85°, always kills.	
Bonhoff, 1892	60°, 20 minutes.	50°, 60 minutes.
Grancher and Ledoux-Lebard, 1892	65°, 5 minutes (attenuates) 70°, 1 minute (kills)	
Forster, 1892	60°, 6 hours 95°, momentary	55°, 3 hours 60°, 45 minutes. 80°, momentary.
De Man, 1893	55°, 4 hour 60°, 1 hour	60°, 45 minutes.
Schroeder, 1894	60°, 15 minutes	
Woodhead, 1895.	50°, 12 hours 60°, 8 hours 65°, 45 minutes 70°, 45 minutes 75°, 2½ minutes	90° (results contradictory).
Marshall, 1899	68°, 20 minutes	60°, 10 minutes.
Th. Smith, 1899	60°, 15 to 20 minutes	
Morgenroth, 1900	55°, 3 hours	70°, 10 minutes. 100°, momentary.
Kobrak, 1900.	50°, 4 hours	
Beck, 1900	100°, 3 hours	100° 80°, 30 minutes.
Galtier, 1900		85°, 6 minutes.
Russell and Hastings, 1900	60°, 20 minutes.	
Herr, 1901	65°, 15 minutes.	80°, 5 seconds.
Hesse, 1901	60°, 20 minutes.	
Levy and Bruns, 1901	65°, 15 minutes.	
Barthel and Stenström, 1901		70°, 15 minutes.
Bang, 1902		65°, 15 minutes.
Tjaden, 1903	85°, 1 to 2 minutes	
Rullmann, 1903	65°, 30 minutes	60°, 20 minutes.
Barthel and Stenström, 1904	80°, 1 minute (uncoagulated)	80°, 1 minute (coagulated).
Russell and Hastings, 1904	80°, 1 minute	
Zelenski, 1906		76°, 20 minutes.
Rosenau, 1907	60°, 20 minutes.	

The above tabular statement shows that my results agree with the work of Yersin, Bonhoff, Schroeder, Th. Smith, Russell and Hastings, and Hesse in that 60° C. for twenty minutes and less is sufficient to kill the tubercle bacillus.

I am now conducting a further series of experiments upon the thermal death point of the tubercle bacillus in butter and milk, naturally and artificially infected, and while this work is not yet completed, the indications again are that 60° C. for twenty minutes is quite sufficient to kill the organism under these conditions.

VIABILITY IN DRIED SPUTUM.

There seems to be a general agreement that the tubercle bacillus may remain alive and virulent in dried sputum for several months. Stone's unusual figure of three years does not stand the light of criticism. The main point established, however, is that the tubercle bacillus may survive in sputum after it dries and is reduced to dust, so that its inhalation may be a danger—how much of a danger depends upon other questions not yet solved.

It is well known that when nonspore-bearing bacteria are dried all but a few soon perish; but these few sometimes survive for unusually long periods of time. This rule probably holds in the case of the tubercle bacillus, but unfortunately we have no quantitative method of determining the number of tubercle bacilli remaining alive. The survival of a few bacilli which are virulent for experimental animals when injected in enormous quantities (as is usually done in laboratory work) would not necessarily mean that human beings are in very great danger from such material under natural conditions.

The following is a tabular abstract of the literature upon the viability of the tubercle bacillus dried in sputum:

Dried tuberculous sputum under different conditions.

Author.	Conditions.	Not killed.	Killed.
Villamin, 1869	Dried tuberculous sputum	Several hours.	
Koch, 1882	do	8 weeks	
Malassez and Vignal, 1883	Alternate drying and moistening eight times of tuberculous sputum.	12 days	
Schill and Fischer, 1884	Sputum containing tubercle bacilli with "spores" dried on glass plates.	126 days	179 days.
	Same, without "spores"	186 days	226 days.
De Thoma, 1886	Dried tuberculous sputum		10 months.
Sormani, 1886	do	2 months	Virulence decreases after 2 months.
	Same, on linen	6 months	Virulence lost at end of 4 months.
Cadeac and Malet, 1888	Pieces dried tuberculous lung on paper exposed in laboratory.	45 days	102 days.
	Dried tuberculous lung allowed to decompose in outer air.	76 days	
	do	80 days	
	do	150 days	After 150 days.
De Souza, 1889	Dried walls of cavities insufflated; caused tuberculosis in 12 of 14 guinea pigs; time of drying not stated.		
Galtier, 1889	Dried tuberculous "material" exposed to air and light at 30° C.	38 days	After 50 days.
	Same, at room temperature	30 days	Do.
Feltz, 1890	Dried tuberculous sputum in road dust, exposed to weather	Over 7 months	
	Same, exposed to sun	About 140 days	
	Dried sputum in rooms	7 to 9 months	
Sawizky, 1891	Tuberculous sputum dried under ordinary conditions of living rooms.	2½ months.	
Stone, 1891	Dried tuberculous sputum	3 years.	
Ransome and Delepine, 1894	Tuberculous sputum exposed in watch glasses to:		
	(*a*) Air and light 4 days, then 15 days in the dark.	19 days.	
	(*b*) Air and darkness 8 days, then 11 days in darkness.		19 days.
	(*c*) Close cupboard 19 days	19 days.	

Dried tuberculous sputum [illegible]—Continued.

Author	Condition	Not killed	Killed
[illegible]	[illegible]		[illegible]
[illegible]	[illegible] exposed to air and light	[illegible] [illegible] [illegible]	4 days 6 days 10 days
	[illegible]		4 days
[illegible]	[illegible]	[illegible]	[illegible]
	In paraffined bottles in dark moist box.	170 days	188 days
	[illegible]	160 days	188 days
	In paraffined bottles in diffused light	124 days...	[illegible]
	In paraffined bottles in thermostat..	[illegible]	100 days
	Cotton stoppered bottles, in dark moist box.	[illegible]	172 days
	Cotton stoppered bottles, in dark closet	[illegible]	[illegible]
	[illegible]	[illegible]	153 days
	[illegible]	[illegible]	110 days
	On towel	70 days	110 days
	On carpet	[illegible]	70 days
	In sand, in moist light place........	12 days	118 days
	[illegible] dry light place	[illegible]	70 days
	In open bottles, out doors, winter ...	110 days	[illegible]
[illegible]	Dried tuberculous excretion at 35°C....	[illegible]	
Rickards, Slack, and Arms 1908	Dried tuberculous sputum in tenement house:		
	(*a*) Sunlight and dry...............	Experiments under way	
	(*b*) Diffused light and dry	1 month	
	(*c*) Dark and dry	[illegible] days...	
	(*d*) Dark and damp basement.	Experiments under way	

THE EFFECT OF SUNLIGHT.

Koch's dictum that direct sunlight kills the tubercle bacillus within a few minutes to several hours has been confirmed by all workers except Feltz. The time required depends upon the brightness of the ray, the time of the year, the latitude, the temperature, the thickness and opacity of the layers, the medium in which the bacilli are embedded, and other conditions.

This action of light corresponds to the well-known germicidal power of the ultra violet rays of the spectrum upon other nonspore-bearing bacteria, and must play an important rôle in diminishing the danger from tuberculous sputum and dust out of doors and in sunny rooms.

The following table gives the data in brief upon the subject:

Effect of direct sunlight upon the tubercle bacillus.

Author	Condition	Not killed	Killed
Koch, 1890	Tubercle bacilli.		Few minutes to several hours.
Feltz, 1890	Tuberculous sputum mixed with dust exposed to sun.	About 10 days.	
Ransome and Delepine, 1894	Pure culture dried on paper in thin layers.		12½ hours.
Migneco, 1895	Tuberculous sputum on linen and woolen cloth.		24 to 30 hours. virulence diminishes in 10 to 15 hours.
Gardiner, 1898	Tuberculous sputum on:		
	Sand	1½ hours	
	Stone	2 hours 5 minutes	
	Wood	24 hours (localized tuberculosis).	
Mitchell and Crouch, 1900	Tuberculous sputum placed under sterilized glass.	35 hours; virulence diminished after 20 hours.	45 hours.
Jousset, 1900	Tuberculous sputum, exposed to dust and sunlight.		4 hours.
Annet, 1903	Dried muco-purulent sputum in small masses about size of one expectoration.	2 to 24 hours.	48 hours.
Cadéac, 1905	Tuberculous sputum on a board		24 hours
	Tuberculous sputum on glass plates exposed to artificial light.	24 hours.	48 hours
Twichell, 1905	Tuberculous sputum exposed to direct sun rays.	1 hour.	7 hours
Di Donna, 1907	Pure cultures	6 days	8 days
Koch, 1890	Cultures of tubercle bacilli exposed to dispersed daylight near window.		5 to 7 days.
Ransome and Delepine, 1894	Pure culturers dried on paper in thin layers, exposed to air and light.		4 days
	Same exposed to sunlight		12¼ hours.

THE TUBERCLE BACILLUS IN WATER.

The tubercle bacillus may probably live and remain virulent in water for several months. We have indications of this in the work of Straus and Dubarry, 1899; Muschold, 1900; Galtier, 1889; Chantemesse and Widal, 1888; and Cadéac and Malet, 1888.

Since the danger of ingesting the tubercle bacillus is now well established, its presence in drinking water assumes a special significance. Tuberculous cattle, as well as tuberculous individuals, discharge large numbers of tubercle bacilli in their dejecta. Sewage-polluted water used for drinking purposes may, therefore, harbor dangers other than the intestinal order of diseases, such as typhoid, cholera, etc. Dixon found acid-fast organisms morphologically resembling the tubercle bacillus in the sewage from hospitals in Philadelphia. Sedgwick and MacNutt,[a] 1908, have examined the theorem first enunciated by Hiram F. Mills and others, but first definitely formulated and published by Allen Hazen, that "for every death from typhoid fever avoided by the purification of public water supplies two or three deaths are avoided from other causes." Sedgwick and MacNutt studied the influence of the purification of polluted water supplies in Lowell and Lawrence, Mass., compared with similar data for Manchester, N. H. The theorem was proved true not only for the cities mentioned, but also for certain other cities, including Hamburg, Germany. Among the causes other than typhoid fever for which the death rates are diminished pulmonary tuberculosis, pneumonia, and infant mortality are prominent. The occurrence and the viability of the tubercle bacillus in water, therefore, becomes a fertile field for research.

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[a] Sedgwick, W. T., and MacNutt, Scott. Typhoid fever and the purification of public water supplies. Science, vol. 27, Aug. 14, 1908, p. 215.

The following is a brief abstract of the work done to date upon the subject:

Tubercle bacillus in water, putrefaction and miscellaneous.

Author.	Condition.	Not killed.	Killed.
[illegible], 188[illegible]	Tubercle bacillus in water	12 months attenuated	
Straus and Dubarry, 1889	Tubercle cultures in water of the river [illegible]:		
	At 20° C	[illegible] days	
	At 28° C	[illegible] days	
	At 35° C	[illegible] days	
	Tubercle cultures in distilled water:		
	At [illegible] C	21 days	
	At 28° C	115 days	
	At [illegible] C	25 days	
Galtier, 1889	Tuberculous spleen in water, 3° to 8° C	17 days	
	Tuberculous products of pigs and cows in running and stagnant water; 17°, 13°, 0° C.	[illegible] days	
	Tuberculous products of cow in running water; 4° to 10° C.	[illegible] months	
[illegible], 1900	Tuberculous sputum in water, sewage, etc.	A number of months.	
	In sewage polluted water	[illegible] months	
Cadéac and Malet, 1888	Tuberculous lung buried.	7[illegible] days	
		121 days	
		159 days	
		167 days	Results negative after 167 days.
	Fragment of tuberculous lung in running water	[illegible] days	
	Piece of tuberculous lung in bowl of water exposed on outer window sill	[illegible] days	
	Piece of tuberculous lung in bowl of water exposed on outer window sill	120 days	150 days.
	[illegible] diluted with water exposed to [illegible]	[illegible]	67 days.
[illegible]	[illegible]	[illegible] months	[illegible] months
[illegible], 1898	Tuberculous sputum, kept fluid in well-corked bottle.		[illegible] months
[illegible], 1905	Dust from papers and books infected with fine spray containing tubercle bacilli.	[illegible] days	[illegible] month
	Sputum dust	[illegible]	[illegible] month
	Cotton fiber containing tubercle bacilli	[illegible]	[illegible] months
	Street dust containing tubercle bacilli.	[illegible]	[illegible] months
[illegible], 1899	[illegible]	[illegible] months	
[illegible], 1899	[illegible]	[illegible]	

SUMMARY.

We have no easy method of determining the death of the tubercle bacillus: its virulence fades before it dies. The criterion of death depends upon animal experimentation.

The tubercle bacillus may be classed with the nonspore-bearing organisms so far as its viability is concerned.

It is doubtful whether the waxy substance protects the bacillus against external harmful influences to any unusual extent.

The thermal death point is 60° C. for twenty minutes. This is much less than was once considered.

Further work upon the viability of the dried tubercle bacillus in dust, sputum, etc., may change our views as to its hardiness and danger under these conditions.

The comparative short life upon artificial culture media contrasts strangely with the long life claimed for it under unfavorable conditions.

Failure to recognize the lesions produced by the dead tubercle bacillus is responsible for some of the false conclusions reached by experimenters upon this subject.

In all cases where the lesions in the test animal are doubtful, inoculations into secondary animals are necessary to determine whether we are dealing with living or dead tubercle bacilli.

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HYGIENIC LABORATORY BULLETINS OF THE PUBLIC HEALTH AND MARINE-HOSPITAL SERVICE.

The Hygienic Laboratory was established in New York, at the Marine Hospital on Staten Island, August, 1887. It was transferred to Washington, with quarters in the Butler Building, June 11, 1891, and a new laboratory building, located in Washington, was authorized by act of Congress, March 3, 1901.

The following *bulletins* [Bulls. Nos. 1-7, 1900 to 1902, Hyg. Lab., U. S. Mar.-Hosp. Serv., Wash.] have been issued:

*No. 1.—Preliminary note on the viability of the *Bacillus pestis*. By M. J. Rosenau.

No. 2.—Formalin disinfection of baggage without apparatus. By M. J. Rosenau.

*No. 3.—Sulphur dioxid as a germicidal agent. By H. D. Geddings.

*No. 4.—Viability of the *Bacillus pestis*. By M. J. Rosenau.

No. 5.—An investigation of a pathogenic microbe (*B. typhi murium* Danyz) applied to the destruction of rats. By M. J. Rosenau.

*No. 6.—Disinfection against mosquitoes with formaldehyde and sulphur dioxid. By M. J. Rosenau.

No. 7.—Laboratory technique: Ring test for indol, by S. B. Grubbs and Edward Francis; Collodium sacs, by S. B. Grubbs and Edward Francis; Microphotography with simple apparatus, by H. B. Parker.

By act of Congress approved July 1, 1902, the name of the "United States Marine-Hospital Service" was changed to the "Public Health and Marine-Hospital Service of the United States," and three new divisions were added to the Hygienic Laboratory.

Since the change of name of the Service the bulletins of the Hygienic Laboratory have been continued in the same numerical order, as follows:

*No. 8. Laboratory course in pathology and bacteriology. By M. J. Rosenau (Revised edition, March, 1904.)

*No. 9. Presence of tetanus in commercial gelatin. By John F. Anderson.

No. 10. Report upon the prevalence and geographic distribution of hookworm disease (uncinariasis or anchylostomiasis) in the United States. By Ch. Wardell Stiles.

*No. 11. An experimental investigation of *Trypanosoma lewisi*. By Edward Francis.

*No. 12. The bacteriological impurities of vaccine virus; an experimental study. By M. J. Rosenau.

*No. 13. A statistical study of the intestinal parasites of 500 white male patients at the United States Government Hospital for the Insane; by Philip E. Garrison, Brayton H. Ransom, and Earle C. Stevenson. A parasitic roundworm (*Agamomermis culicis* n. g., n. sp.) in American mosquitoes (*Culex sollicitans*); by Ch. Wardell Stiles. The type species of the cestode genus *Hymenolepis;* by Ch. Wardell Stiles.

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No. 17. Illustrated key to the trematode parasites of man. By Ch. Wardell Stiles.

*No. 18. An account of the tapeworms of the genus *Hymenolepis* parasitic in man, including reports of several new cases of the dwarf tapeworm (*H. nana*) in the United States. By Brayton H. Ransom.

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*No. 20.—A zoological investigation into the cause, transmission, and source of Rocky Mountain "spotted fever." By Ch. Wardell Stiles.

No. 21.—The immunity unit for standardizing diphtheria antitoxin (based on Ehrlich's normal serum). Official standard prepared under the act approved July 1, 1902. By M. J. Rosenau.

*No. 22.—Chloride of zinc as a deodorant, antiseptic, and germicide. By T. B. McClintic.

*No. 23.—Changes in the Pharmacopœia of the United States of America. Eighth Decennial Revision. By Reid Hunt and Murray Galt Motter.

No. 24.—The International Code of Zoological Nomenclature as applied to medicine. By Ch. Wardell Stiles.

No. 25.—Illustrated key to the cestode parasites of man. By Ch. Wardell Stiles.

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*No. 35.—Report on the origin and prevalence of typhoid fever in the District of Columbia. By M. J. Rosenau, L. L. Lumsden, and Joseph H. Kastle. (Including articles contributed by Ch. Wardell Stiles, Joseph Goldberger, and A. M. Stimson.)

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A reexamination of the original specimen of *Taenia saginata abietina* (Weinland, 1858), by Ch. Wardell Stiles and Joseph Goldberger.

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